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AMERICAN SOCIETY FOR CIRCUMPOLAR HEALTH

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Effective control of fasting and postprandial glucose—patient after patient, meal after meal, year after year.

Insulin when it's needed

Insulin levels are rapidly elevated in response to a meal, then return promptly to basal levels after the meal challenge subsides.

Timed to minimize risks

Rapidly metabolized and excreted, with an excellent safety profile.[1] As with all sulfonylureas, hypoglycemia may occur.

In concert with diet in non-insulin-dependent diabetes mellitus

Glucotrol®

(glipizide) 5-mg and 10-mg Scored Tablets

SYNCHRONIZED SULFONYLUREA THERAPY

Please see brief summary of Glucotrol® (glipizide) prescribing information on next page.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

Reference:
1. Sachs R, Frank M, Fishman SK. Overview of clinical experience with glipizide. In *Glipizide: A Worldwide Review*. Princeton, NJ, Excerpta Medica, 1984, pp 163-172.

GLUCOTROL® (glipizide) Tablets
Brief Summary of Prescribing Information

INDICATIONS AND USAGE: GLUCOTROL is indicated as an adjunct to diet for the control of hyperglycemia in patients with non-insulin-dependent diabetes mellitus (NIDDM, type II) after an adequate trial of dietary therapy has proved unsatisfactory.

CONTRAINDICATIONS: GLUCOTROL is contraindicated in patients with known hypersensitivity to the drug or with diabetic ketoacidosis, with or without coma, which should be treated with insulin.

SPECIAL WARNING ON INCREASED RISK OF CARDIOVASCULAR MORTALITY. The administration of oral hypoglycemic drugs has been reported to be associated with increased cardiovascular mortality as compared to treatment with diet alone or diet plus insulin. This warning is based on the study conducted by the University Group Diabetes Program (UGDP), a long-term prospective clinical trial designed to evaluate the effectiveness of glucose-lowering drugs in preventing or delaying vascular complications in patients with non-insulin-dependent diabetes. The study involved 823 patients who were randomly assigned to one of four treatment groups (*Diabetes*, 19, supp. 2:747-830, 1970).

UGDP reported that patients treated for 5 to 8 years with diet plus a fixed dose of tolbutamide (1.5 grams per day) had a rate of cardiovascular mortality approximately 2-1/2 times that of patients treated with diet alone. A significant increase in total mortality was not observed, but the use of tolbutamide was discontinued based on the increase in cardiovascular mortality, thus limiting the opportunity for the study to show an increase in overall mortality. Despite controversy regarding the interpretation of these results, the findings of the UGDP study provide an adequate basis for this warning. The patient should be informed of the potential risks and advantages of GLUCOTROL and of alternative modes of therapy.

Although only one drug in the sulfonylurea class (tolbutamide) was included in this study, it is prudent from a safety standpoint to consider that this warning may also apply to other oral hypoglycemic drugs in this class, in view of their close similarities in mode of action and chemical structure.

PRECAUTIONS: Renal and Hepatic Disease: The metabolism and excretion of GLUCOTROL may be slowed in patients with impaired renal and/or hepatic function. Hypoglycemia may be prolonged in such patients should it occur.

Hypoglycemia: All sulfonylureas are capable of producing severe hypoglycemia. Proper patient selection, dosage, and instructions are important to avoid hypoglycemia. Renal or hepatic insufficiency may increase the risk of hypoglycemic reactions. Elderly, debilitated or malnourished patients and those with adrenal or pituitary insufficiency are particularly susceptible to the hypoglycemic action of glucose-lowering drugs. Hypoglycemia may be difficult to recognize in the elderly or people taking beta-adrenergic blocking drugs. Hypoglycemia is more likely to occur when caloric intake is deficient, after severe or prolonged exercise, when alcohol is ingested, or when more than one glucose-lowering drug is used.

Loss of Control of Blood Glucose: A loss of control may occur in diabetic patients exposed to stress such as fever, trauma, infection or surgery. It may then be necessary to discontinue GLUCOTROL and administer insulin.

Laboratory Tests: Blood and urine glucose should be monitored periodically. Measurement of glycosylated hemoglobin may be useful.

Information for Patients: Patients should be informed of the potential risks and advantages of GLUCOTROL, of alternative modes of therapy, as well as the importance of adhering to dietary instructions, of a regular exercise program, and of regular testing of urine and/or blood glucose. The risks of hypoglycemia, its symptoms and treatment, and conditions that predispose to its development should be explained to patients and responsible family members. Primary and secondary failure should also be explained.

Drug Interactions: The hypoglycemic action of sulfonylureas may be potentiated by certain drugs including nonsteroidal anti-inflammatory agents and other drugs that are highly protein bound, salicylates, sulfonamides, chloramphenicol, probenecid, coumarins, monoamine oxidase inhibitors, and beta adrenergic blocking agents. *In vitro* studies indicate that GLUCOTROL binds differently than tolbutamide and does not interact with salicylate or dicumarol. However, caution must be exercised in extrapolating these findings to a clinical situation. Certain drugs tend to produce hyperglycemia and may lead to loss of control, including the thiazides and other diuretics, corticosteroids, phenothiazines, thyroid products, estrogens, oral contraceptives, phenytoin, nicotinic acid, sympathomimetics, calcum channel blocking drugs, and isoniazid. A potential interaction between oral miconazole and oral hypoglycemic agents leading to severe hypoglycemia has been reported. Whether this interaction also occurs with the intravenous, topical, or vaginal preparations of miconazole is not known.

Carcinogenesis, Mutagenesis, Impairment of Fertility: A 20-month study in rats and an 18-month study in mice at doses up to 75 times the maximum human dose revealed no evidence of drug-related carcinogenicity. Bacterial and *in vivo* mutagenicity tests were uniformly negative. Studies in rats of both sexes at doses up to 75 times the human dose showed no effects on fertility.

Pregnancy: Pregnancy Category C: GLUCOTROL (glipizide) was found to be mildly fetotoxic in rat reproductive studies at all dose levels (5-50 mg/kg). This fetotoxicity has been similarly noted with other sulfonylureas, such as tolbutamide and tolazamide. The effect is perinatal and believed to be directly related to the pharmacologic (hypoglycemic) action of GLUCOTROL. In studies in rats and rabbits no teratogenic effects were found. There are no adequate and well controlled studies in pregnant women. GLUCOTROL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Because recent information suggests that abnormal blood glucose levels during pregnancy are associated with a higher incidence of congenital abnormalities, many experts recommend that insulin be used during pregnancy to maintain blood glucose levels as close to normal as possible.

Nonteratogenic Effects: Prolonged severe hypoglycemia has been reported in neonates born to mothers who were receiving a sulfonylurea drug at the time of delivery. This has been reported more frequently with the use of agents with prolonged half-lives. GLUCOTROL should be discontinued at least one month before the expected delivery date.

Nursing Mothers: Since some sulfonylurea drugs are known to be excreted in human milk, insulin therapy should be considered if nursing is to be continued.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: In controlled studies, the frequency of serious adverse reactions reported was very low. Of 702 patients, 11.8% reported adverse reactions and in only 1.5% was GLUCOTROL discontinued.

Hypoglycemia: See PRECAUTIONS and OVERDOSAGE sections.

Gastrointestinal: Gastrointestinal disturbances, the most common, were reported with the following approximate incidence: nausea and diarrhea, one in 70; constipation and gastralgia, one in 100. They appear to be dose-related and may disappear on division or reduction of dosage. Chloestatic jaundice may occur rarely with sulfonylureas. GLUCOTROL should be discontinued if this occurs.

Dermatologic: Allergic skin reactions including erythema, morbilliform or maculopapular eruptions, urticaria, pruritus, and eczema have been reported in about one in 70 patients. These may be transient and may disappear despite continued use of GLUCOTROL; if skin reactions persist, the drug should be discontinued. Porphyria cutanea tarda and photosensitivity reactions have been reported with sulfonylureas.

Hematologic: Leukopenia, agranulocytosis, thrombocytopenia, hemolytic anemia, aplastic anemia, and pancytopenia have been reported with sulfonylureas.

Metabolic: Hepatic porphyria and disulfiram-like alcohol reactions have been reported with sulfonylureas. Clinical experience to date has shown that GLUCOTROL has an extremely low incidence of disulfiram-like reactions.

Endocrine Reactions: Cases of hyponatremia and the syndrome of inappropriate antidiuretic hormone (SIADH) secretion have been reported with this and other sulfonylureas.

Miscellaneous: Dizziness, drowsiness, and headache have been reported in about one in fifty patients treated with GLUCOTROL. They are usually transient and seldom require discontinuance of therapy.

OVERDOSAGE: Overdosage of sulfonylureas including GLUCOTROL can produce hypoglycemia. If hypoglycemic coma is diagnosed or suspected, the patient should be given a rapid intravenous injection of concentrated (50%) glucose solution. This should be followed by a continuous infusion of a more dilute (10%) glucose solution at a rate that will maintain the blood glucose at a level above 100 mg/dL. Patients should be closely monitored for a minimum of 24 to 48 hours since hypoglycemia may recur after apparent clinical recovery. Clearance of GLUCOTROL from plasma would be prolonged in persons with liver disease. Because of the extensive protein binding of GLUCOTROL (glipizide), dialysis is unlikely to be of benefit.

DOSAGE AND ADMINISTRATION: There is no fixed dosage regimen for the management of diabetes mellitus with GLUCOTROL; in general, it should be given approximately 30 minutes before a meal to achieve the greatest reduction in postprandial hyperglycemia.

Initial Dose: The recommended starting dose is 5 mg before breakfast. Geriatric patients or those with liver disease may be started on 2.5 mg. Dosage adjustments should ordinarily be in increments of 2.5-5 mg, as determined by blood glucose response. At least several days should elapse between titration steps.

Maximum Dose: The maximum recommended total daily dose is 40 mg.

Maintenance: Some patients may be effectively controlled on a once-a-day regimen, while others show better response with divided dosing. Total daily doses above 15 mg should ordinarily be divided.

HOW SUPPLIED: GLUCOTROL is available as white, dye-free, scored diamond-shaped tablets imprinted as follows: 5 mg tablet—Pfizer 411 (NDC 5 mg 0049-4110-66) Bottles of 100; 10 mg tablet—Pfizer 412 (NDC 10 mg 0049-4120-65) Bottles of 100.

CAUTION: Federal law prohibits dispensing without prescription.

More detailed professional information available on request.

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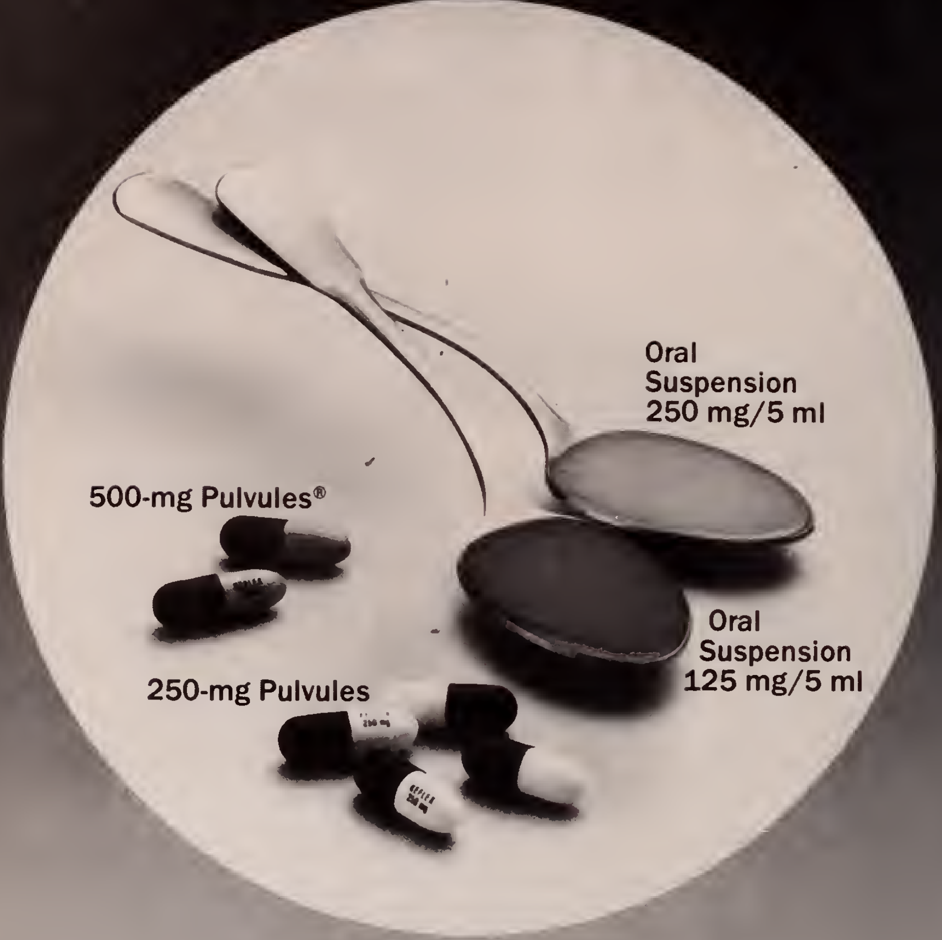

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Every day more and more physicians are hearing something remarkable from some of their hypertensive patients...

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from the ones on once-daily INDERAL® LA (PROPRANOLOL HCl)

with a side-effect profile unsurpassed by atenolol or metoprolol.

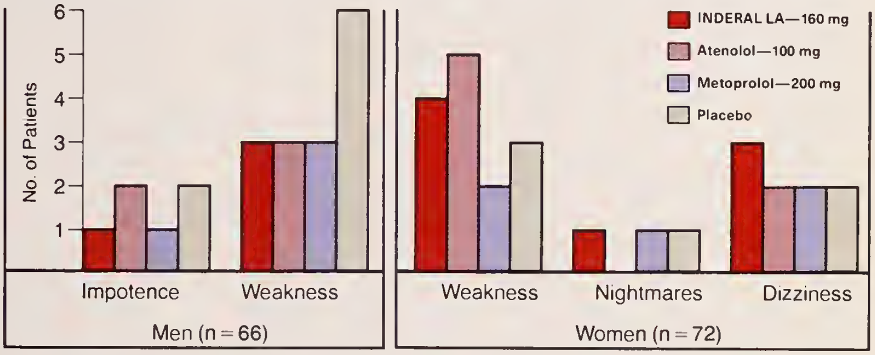
As seen in this double-blind, crossover, placebo-controlled study.[1]

Which shows you how truly well tolerated once-daily INDERAL LA can be.

What comes as *no* surprise, of course, is that it gives you the antihypertensive effectiveness you've come to expect from INDERAL.

Selected Side Effects

INDERAL LA as well tolerated as atenolol and metoprolol in a double-blind, crossover, placebo-controlled study of 138 hypertensives[1]



INDERAL® LA. For control.
Comfortable control. Once a day.
It's the last word.

Hypertensives: Feeling well and doing well, all in one.

INDERAL® LA
(PROPRANOLOL HCl)
LONG ACTING CAPSULES

or

INDERIDE® LA
(PROPRANOLOL HCl [INDERAL® LA]/ HYDROCHLOROTHIAZIDE)
LONG ACTING CAPSULES

As with all fixed-combination antihypertensives, INDERIDE LA is not indicated for the initial treatment of hypertension.

INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

Please turn page for brief summary of prescribing information

Feeling well and doing well, all in one.

ONCE-DAILY LONG ACTING CAPSULES 80 mg 120 mg 160 mg

INDERAL® LA
(PROPRANOLOL HCl)

ONCE-DAILY LONG ACTING CAPSULES 80/50 120/50 160/50

INDERIDE® LA
Each capsule contains propranolol HCl (INDERAL® LA), 80 mg, 120 mg, or 160 mg, and hydrochlorothiazide, 50 mg

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULARS.)*

INDERAL® LA Brand of PROPRANOLOL HYDROCHLORIDE **(Long Acting Capsules)**
INDERIDE® LA Brand of PROPRANOLOL HYDROCHLORIDE (INDERAL® LA) and HYDROCHLOROTHIAZIDE **(Long Acting Capsules)**

INDERAL LA and INDERIDE LA Capsules should not be considered simple mg-for-mg substitutes for INDERAL and INDERIDE Tablets Please see package circulars

CONTRAINDICATIONS

Propranolol hydrochloride (INDERAL® LA): Propranolol is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with propranolol

Hydrochlorothiazide: Hydrochlorothiazide is contraindicated in patients with anuria or hypersensitivity to this or other sulfonamide-derived drugs

WARNINGS

Propranolol hydrochloride (INDERAL® LA): CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated, and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or propranolol should be discontinued (gradually if possible).

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction following *abrupt* discontinuance of propranolol therapy Therefore, when discontinuance of propranolol is planned the dosage should be gradually reduced and the patient carefully monitored In addition, when propranolol is prescribed for angina pectoris, the patient should be cautioned against interruption or cessation of therapy without the physician's advice If propranolol therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute propranolol therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

THYROTOXICOSIS. Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)— PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD, IN GENERAL, NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution, since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

DIABETES AND HYPOGLYCEMIA. Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes In these patients, it may be more difficult to adjust the dosage of insulin Hypoglycemic attacks may be accompanied by a precipitous elevation of blood pressure

Hydrochlorothiazide: Thiazides should be used with caution in severe renal disease In patients with renal disease, thiazides may precipitate azotemia In patients with impaired renal function, cumulative effects of the drug may develop.

Thiazides should also be used with caution in patients with impaired hepatic function or progressive liver disease since minor alterations of fluid and electrolyte balance may precipitate hepatic coma

Thiazides may add to or potentiate the action of other antihypertensive drugs Potentiation occurs with ganglionic or peripheral adrenergic-blocking drugs

Sensitivity reactions may occur in patients with a history of allergy or bronchial asthma

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported

PRECAUTIONS

Propranolol hydrochloride (INDERAL® LA): GENERAL Propranolol should be used with caution in patients with impaired hepatic or renal function Propranolol is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that propranolol may interfere with the glaucoma screening test Withdrawal may lead to a return of increased intraocular pressure

CLINICAL LABORATORY TESTS Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs, such as reserpine, should be closely observed if propranolol is administered The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks or orthostatic hypotension

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential In 18-month studies, in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity There were no drug-related tumorigenic effects at any of the dosage levels Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug

PREGNANCY Pregnancy Category C Propranolol has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximal recommended human dose There are no adequate and well-controlled studies in pregnant women Propranolol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

NURSING MOTHERS Propranolol is excreted in human milk Caution should be exercised when propranolol is administered to a nursing mother

PEDIATRIC USE Safety and effectiveness in children have not been established

Hydrochlorothiazide: GENERAL Periodic determination of serum electrolytes to detect possible electrolyte imbalance should be performed at appropriate intervals

All patients receiving thiazide therapy should be observed for clinical signs of fluid or electrolyte imbalance, namely Hyponatremia, hypochloremic alkalosis and hypokalemia Serum and urine electrolyte determinations are particularly important when the patient is vomiting excessively or receiving parenteral fluids Medication such as digitalis may also influence serum electrolytes Warning signs irrespective of cause are Dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue hypotension, oliguria tachycardia and gastrointestinal disturbances such as nausea and vomiting

Hypokalemia may develop, especially with brisk diuresis, when severe cirrhosis is present, or during concomitant use of corticosteroids or ACTH

Interference with adequate oral electrolyte intake will also contribute to hypokalemia Hypokalemia can sensitize or exaggerate the response of the heart to the toxic effect of digitalis (eg, increased ventricular irritability) Hypokalemia may be avoided or treated by use of potassium supplements, such as foods with a high potassium content

Any chloride deficit is generally mild and usually does not require specific treatment except under extraordinary circumstances (as in liver or renal disease) Dilutional hyponatremia may occur in edematous patients in hot weather appropriate therapy is water restriction, rather than administration of salt, except in rare instances when the hyponatremia is life-threatening In actual salt depletion appropriate replacement is the therapy of choice

Hyperuricemia may occur or frank gout may be precipitated in certain patients receiving thiazide therapy

Insulin requirements in diabetic patients may be increased, decreased, or unchanged Diabetes mellitus which has been latent may become manifest during thiazide administration

If progressive renal impairment becomes evident, consider withholding or discontinuing diuretic therapy

Thiazides may decrease serum PBI levels without signs of thyroid disturbance

Calcium excretion is decreased by thiazides Pathologic changes in the parathyroid gland with hypercalcemia and hypophosphatemia have been observed in a few patients on prolonged thiazide therapy The common complications of hyperparathyroidism, such as renal lithiasis, bone resorption, and peptic ulceration have not been seen Thiazides should be discontinued before carrying out tests for parathyroid function

DRUG INTERACTIONS Thiazide drugs may increase the responsiveness to tubocurarine

The antihypertensive effects of thiazides may be enhanced in the postsympathectomy patient Thiazides may decrease arterial responsiveness to norepinephrine This diminution is not sufficient to preclude effectiveness of the pressor agent for therapeutic use

PREGNANCY Pregnancy Category C Thiazides cross the placental barrier and appear in cord blood The use of thiazides in pregnancy requires that the anticipated benefit be weighed against possible hazards to the fetus These hazards include fetal or neonatal jaundice, thrombocytopenia, and possibly other adverse reactions which have occurred in the adult

NURSING MOTHERS Thiazides appear in human milk If use of the drug is deemed essential, the patient should stop nursing

PEDIATRIC USE Safety and effectiveness in children have not been established

ADVERSE REACTIONS

Propranolol hydrochloride (INDERAL® LA): Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy

Cardiovascular Bradycardia, congestive heart failure intensification of AV block hypotension, paresthesia of hands; thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type

Central Nervous System. Lightheadedness; mental depression manifested by insomnia lassitude, weakness, fatigue, reversible mental depression progressing to catatonia visual disturbances hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability slightly clouded sensorium, and decreased performance on neuropsychometrics

Gastrointestinal. Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea constipation; mesenteric arterial thrombosis, ischemic colitis

Allergic: Pharyngitis and agranulocytosis, erythematous rash; fever combined with aching and sore throat, laryngospasm and respiratory distress

Respiratory Bronchospasm.

Hematologic Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported

Miscellaneous. Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes, and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol

Hydrochlorothiazide:

Gastrointestinal Anorexia, gastric irritation, nausea, vomiting, cramping, diarrhea, constipation; jaundice (intrahepatic cholestatic jaundice); pancreatitis sialadenitis

Central Nervous System Dizziness, vertigo, paresthesias, headache, xanthopsia

Hematologic. Leukopenia; agranulocytosis, thrombocytopenia, aplastic anemia

Cardiovascular Orthostatic hypotension (may be aggravated by alcohol, barbiturates or narcotics)

Hypersensitivity Purpura, photosensitivity; rash; urticaria; necrotizing angiitis (vasculitis, cutaneous vasculitis), fever, respiratory distress, including pneumonitis, anaphylactic reactions

Other Hyperglycemia, glycosuria, hyperuricemia muscle spasm weakness, restlessness, transient blurred vision.

Whenever adverse reactions are moderate or severe, thiazide dosage should b reduced or therapy withdrawn

* The appearance of these capsules is a registered trademark of Ayerst Laboratories.

REFERENCE:

1. Ravid M, Lang R, Jutrin I The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily *Arch Intern Med* 1985,145 1321–132

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CONFIRMED BY CLINICAL EVIDENCE

ZANTAC® 150 h.s.

ranitidine HCl/Glaxo 150 mg tablets

EFFECTIVE MAINTENANCE THERAPY

for healed duodenal ulcer patients

CONFIRMED

In two randomized, double-blind, and well-controlled clinical trials, ZANTAC 150 mg h.s. significantly superior to cimetidine 400 mg h.s. for maintenance therapy in healed duodenal ulcers.

Percent of patients with observed duodenal ulcer recurrence

		0-4 months	0-8 months	0-12 months	No. patients
USA[1]	ranitidine 150 mg h.s.	9%	14%*	16%†	60
	cimetidine 400 mg h.s.	23%	34%	43%	66
UK, Ireland, Australia[2]	ranitidine 150 mg h.s.	8%‡	14%‡	23%‡	243
	cimetidine 400 mg h.s.	21%	34%	37%	241

*p=0.02
†p=0.01
‡p≤0.004
%=life-table estimates
All patients were permitted prn antacids for relief of pain.

These two trials used the currently recommended dosing regimen of cimetidine (400 mg h.s.) and ranitidine (150 mg h.s.). A comparison of other dosing regimens has not been studied.

The studied dosing regimens are not equivalent with respect to the degree and duration of acid suppression or suppression of nocturnal acid.

The superiority of ranitidine over cimetidine in these trials indicates that the dosing regimen currently recommended for cimetidine is less likely to be as successful in maintenance therapy.

Convenient once-a-night dose with a low incidence of side effects[3]

Headache, sometimes severe, seems to be related to ranitidine administration. Other side effects have been reported; for a complete listing, see the ADVERSE REACTIONS section in the Brief Summary.

No significant interference with the hepatic cytochrome P-450 enzyme system at recommended doses

ZANTAC 150 mg has no significant drug interactions with theophylline, phenytoin, or warfarin. The bioavailability of certain medications whose absorption is dependent on a low gastric pH may be altered when ZANTAC or other medications that decrease gastric acidity are administered.

Zantac® 150
ranitidine HCl/Glaxo 150 mg tablets

One tablet at bedtime
for maintenance

See next page for references and
Brief Summary of Product Information.

Glaxo/ROCHE®

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Zantac® 150

ranitidine HCl/Glaxo 150 mg tablets

One tablet at bedtime for maintenance therapy in healed duodenal ulcer patients

References:

1. Silvis SE, Griffin J, Hardin R, et al. Final report on the United States multicenter trial comparing ranitidine to cimetidine as maintenance therapy following healing of duodenal ulcer. *J Clin Gastroenterol* 1985;7(6):482-487.
2. Gough KR, Korman MG, Bardhan KD, et al: Ranitidine and cimetidine in prevention of duodenal ulcer relapse: A double-blind, randomised, multicentre, comparative trial. *Lancet* 1984;ii:659-662.
3. Data available on request, Glaxo Inc.

ZANTAC® 150 Tablets
(ranitidine hydrochloride)
ZANTAC® 300 Tablets
(ranitidine hydrochloride)

BRIEF SUMMARY OF PRODUCT INFORMATION

The following is a brief summary only. Before prescribing, see complete prescribing information in ZANTAC® product labeling.

INDICATIONS AND USAGE: ZANTAC® is indicated in:

1. Short-term treatment of **active duodenal ulcer.** Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD).** Symptomatic relief commonly occurs within one or two weeks after starting therapy and is maintained throughout a six-week course of therapy.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

CONTRAINDICATIONS: ZANTAC® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS: Symptomatic response to ZANTAC® therapy does not preclude the presence of gastric malignancy.

Since ZANTAC is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see **DOSAGE AND ADMINISTRATION**). Caution should be observed in patients with hepatic dysfunction since ZANTAC is metabolized in the liver.

False-positive tests for urine protein with Multistix® may occur during ZANTAC therapy, and therefore testing with sulfosalicylic acid is recommended.

Although recommended doses of ZANTAC do not inhibit the action of cytochrome P-450 enzymes in the liver, there have been isolated reports of drug interactions which suggest that ZANTAC may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Lack of experience to date precludes recommending ZANTAC for use in children or pregnant patients. Since ZANTAC is secreted in human milk, caution should be exercised when administered to a nursing mother.

ADVERSE REACTIONS: Headache, sometimes severe, seems to be related to ZANTAC® administration. Constipation, diarrhea, nausea/vomiting, and abdominal discomfort/pain have been reported. There have been rare reports of malaise, dizziness, somnolence, insomnia, vertigo, tachycardia, bradycardia, premature ventricular beats, and arthralgias. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients.

In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid IV for seven days, and in 4 of 24 subjects receiving 50 mg qid for five days. With oral administration there have been occasional reports of reversible hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice.

There have been rare reports of reversible leukopenia, granulocytopenia, thrombocytopenia, and pancytopenia.

Although controlled studies have shown no antiandrogenic activity, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving ZANTAC, but the incidence did not differ from that in the general population.

Incidents of rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia, have been reported, as well as rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia) and small increases in serum creatinine.

OVERDOSAGE: Information concerning possible overdosage and its treatment appears in the full prescribing information.

DOSAGE AND ADMINISTRATION. Active Duodenal Ulcer: The current recommended adult oral dosage is 150 mg twice daily. An alternate dosage of 300 mg once daily at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated.

Maintenance Therapy: The current recommended adult oral dosage is 150 mg at bedtime.

Pathological Hypersecretory Conditions (such as Zollinger-Ellison Syndrome): The current recommended adult oral dosage is 150 mg twice a day. In some patients it may be necessary to administer ZANTAC 150-mg doses more frequently. Doses should be adjusted to individual patient needs, and should continue as long as clinically indicated. Doses up to 6 g/day have been employed in patients with severe disease.

Benign Gastric Ulcer: The current recommended adult oral dosage is 150 mg twice a day.

GERD: The current recommended adult oral dosage is 150 mg twice a day.

Dosage Adjustment for Patients with Impaired Renal Function: On the basis of experience with a group of subjects with severely impaired renal function treated with ZANTAC, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

HOW SUPPLIED: ZANTAC® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) and unit dose packs of 100 tablets (NDC 0173-0393-47).

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Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.

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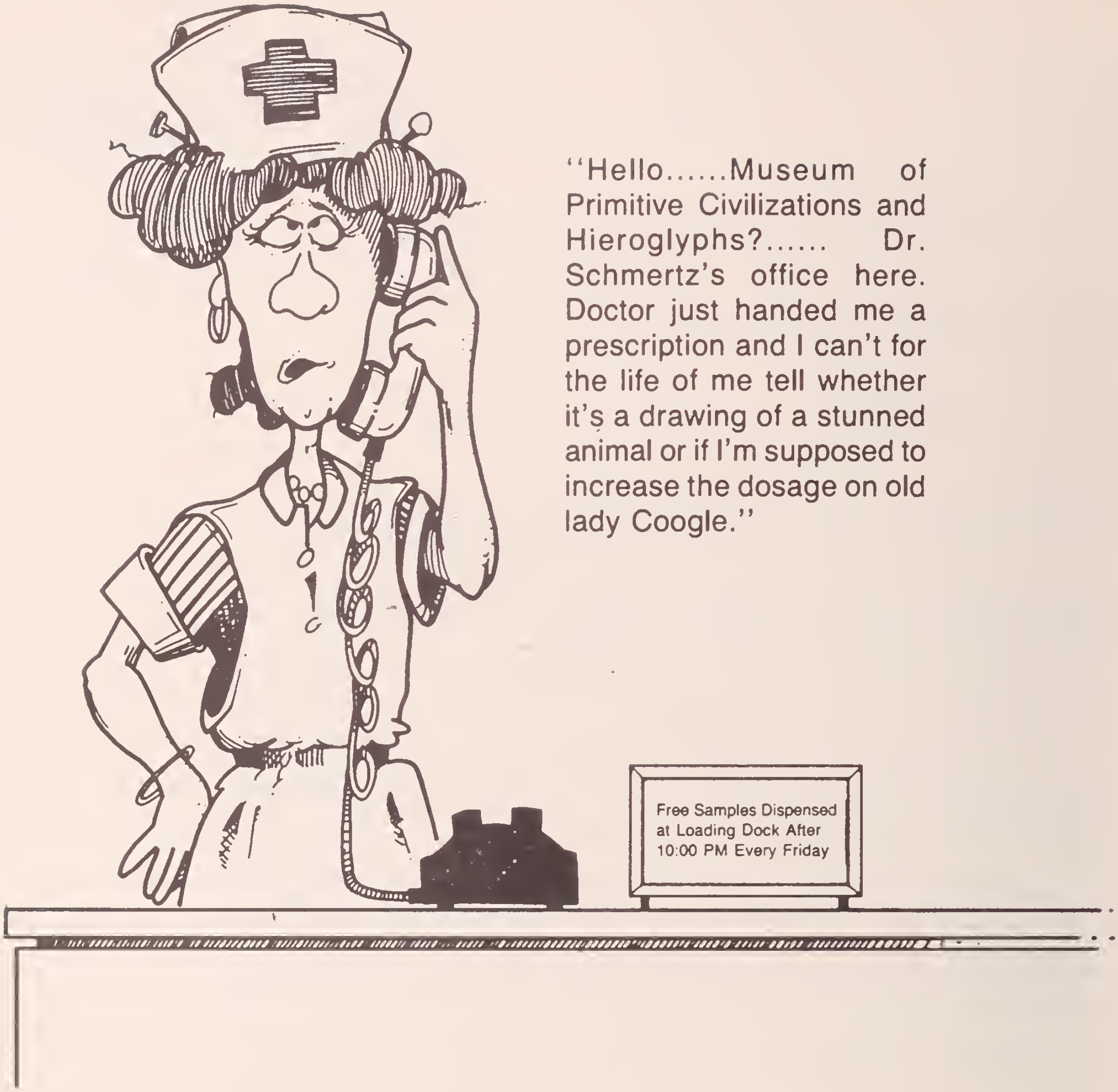

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Alaska State Hepatitis B Program— Past, Present and Future

by Elizabeth A. Tower, M.D., M.P.H.[1]

As of October 1986, 20% of Alaska's 525,000 people were tested for past or present evidence of infection with the hepatitis B virus. More than 30,000 susceptible individuals considered at high risk of acquiring hepatitis B have completed the three-dose series of hepatitis B vaccine. The screening and vaccination program, a joint venture of the State of Alaska Division of Public Health, Alaska Native Health Service, and Centers for Disease Control Arctic Investigations Laboratory (AIL-CDC), succeeded in spite of the high cost of hepatitis B vaccine and formidable logistic problems.

Diminished State revenues have now necessitated considerable reduction in the ability of the Alaska Division of Public Health to continue direct funding of the program. The purpose of this article is to review the accomplishments of the three-year program from the state perspective and to present suggestions that may aid private physicians and other health care providers in cost-effective hepatitis B control measures.

BACKGROUND

A high prevalence of hepatitis B infection in several southwestern Alaska Eskimo villages was first observed in the early 1970's (1). By 1980 extensive epidemiological studies had established the high prevalence of infection in some neighboring villages as well. Several young Yupik Eskimos succumbed to hepatitis B-related vasculitis and hepatocellular carcinoma (2). In 1981 and 1982, AIL-CDC conducted a field demonstration project using the newly licensed hepatitis B vaccine in 14 southwestern Alaska villages, demonstrating that the vaccine could be administered safely and effectively in rural Alaska (3). Pleas to Alaska's Congressional delegation in 1983 resulted in an initial federal appropriation of $1,000,000 to assess the extent of the problem in the Native population and to vaccinate those at the highest risk. The Alaska Hepatitis B Control Program was born.

In February 1983 the Alaska Native Health Service, by memorandum of agreement, designated the State of Alaska, Division of Public Health, with its laboratory capability and statewide cadre of public health nurses experienced in immunization programs, to administer the screening and vaccination effort. An additional $500,000, provided by the Alaska State Legislature in early 1983, enabled the Division of Public Health to include all Alaskans in the control program.

The Northern Regional Laboratory, Section of Laboratories, Division of Public Health, had extensive experience in Enzyme Immunoassay (ELISA) testing (4) when the first comprehensive village screening started in March 1983. The ELISA test for hepatitis B anti-core antibody (HBcAb), the lifelong marker of hepatitis B virus (HBV) infection, was chosen as the initial screening test because a high prevalence of HBV infection was anticipated. One of the goals of the program was the identification of chronic carriers of the virus so that they could be evaluated for evidence of early resectable primary hepatocellular carcinoma (PHC) (5). Sera positive for HBcAb were tested for hepatitis B surface antigen (HBsAg) and antibody (HBsAb) in order to identify carriers and to detect those persons who were already immune. Antigen positive sera were tested for hepatitis B "e" antigen (HBeAg) and "e" antibody (HBeAb) to assess the infectiousness of the carriers (6).

By early 1984, all 54 villages in southwestern Alaska had been serologically screened by phlebotomy teams contracted by the Division of Public Health, and vaccination of susceptible individuals initiated by the State public health nurses. All persons, regardless of race, belonging to designated high-risk groups such as health care workers, male homosexuals, intravenous drug users, institutionalized mentally retarded, and Asian immigrants (7) were also offered screening and vaccination at public health centers throughout the state.

Screening in northern and southeastern Alaska, accomplished during 1984 and early 1985, revealed a prevalence of hepatitis B infection insufficiently high to justify the expense of the comprehensive vaccination program conducted by the state. In order to complete screening and vaccination of all Alaska Natives by 1987, as mandated in the appropriation of additional Indian Health funds, the Alaska Native Health Service contracted with regional Native Health Corporations to continue screening and vaccination of susceptible Alaska Natives.

The Division of Public Health transferred the responsibility for the hepatitis B control from the Section of Communicable Disease Control to the Epidemiology Office late in 1985. Decreased state funding in 1986 has severely restricted the ability of the state to provide hepatitis B vaccine free-of-charge for persons belonging to the recognized high risk groups.

---

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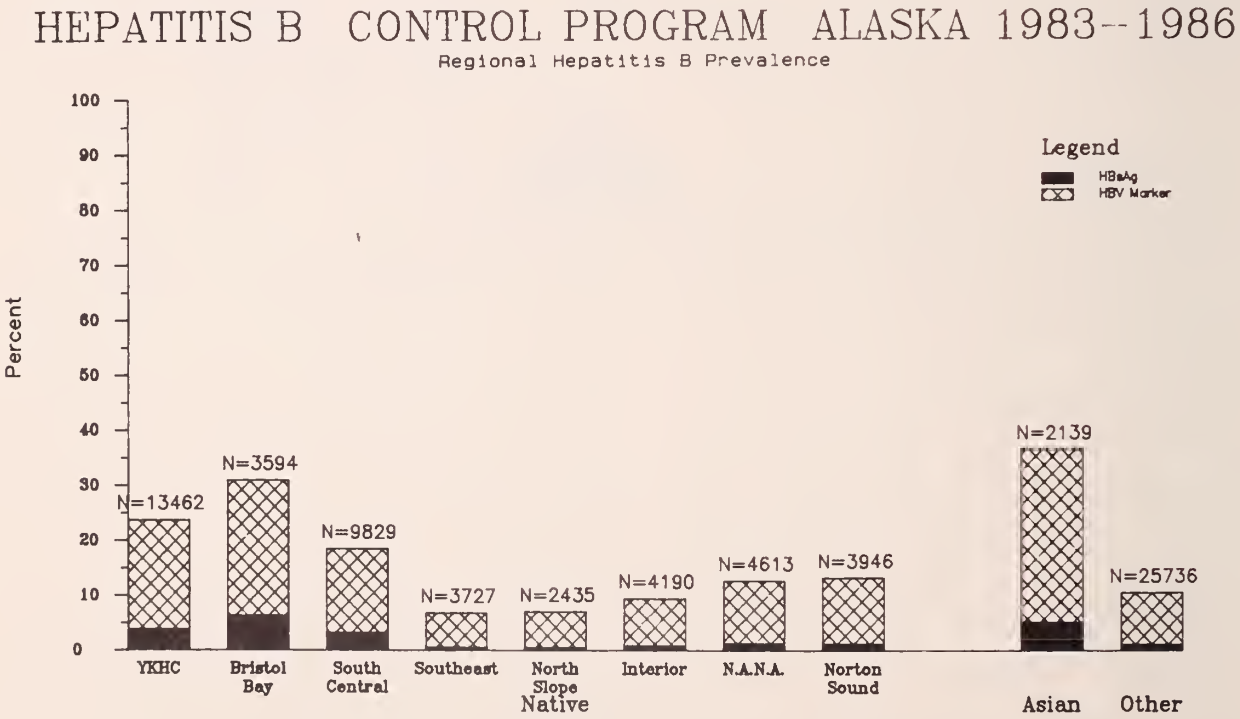
FINDINGS

Hepatitis B infection is not a uniformly serious problem in all areas of Alaska.

The high prevalence of HBV infection was concentrated in the Yukon-Kuskokwim Delta and Bristol Bay regions (Figure 1). Several Lake Iliamna villages adjacent to Bristol Bay and Anchorage accounted for the relatively high prevalence in the southcentral region. A spotty distribution of infection from village to village existed even in the high prevalence areas. Although hard to substantiate retrospectively, some life style factors such as the premastication of food for infants and pregnancy at an early age when chronic carriers are likely to be still in the highly infectious stage, may have facilitated the spread of hepatitis B infection (8).

Figure 1



Unless treated prophylactically with hepatitis B immune globulin (HBIG) and/or hepatitis B vaccine, 90% of infants born to mothers who are HBeAg positive will become chronic carriers of the virus (9). However, vertical transmission has not played an important role in dissemination of disease in Alaska, possibly because most Alaskan Native women of childbearing age are not HBeAg positive. Crowded living conditions and chronic skin infection from insect bites, scabies, and impetigo have probably contributed to the spread of infection among young children in the villages (10).

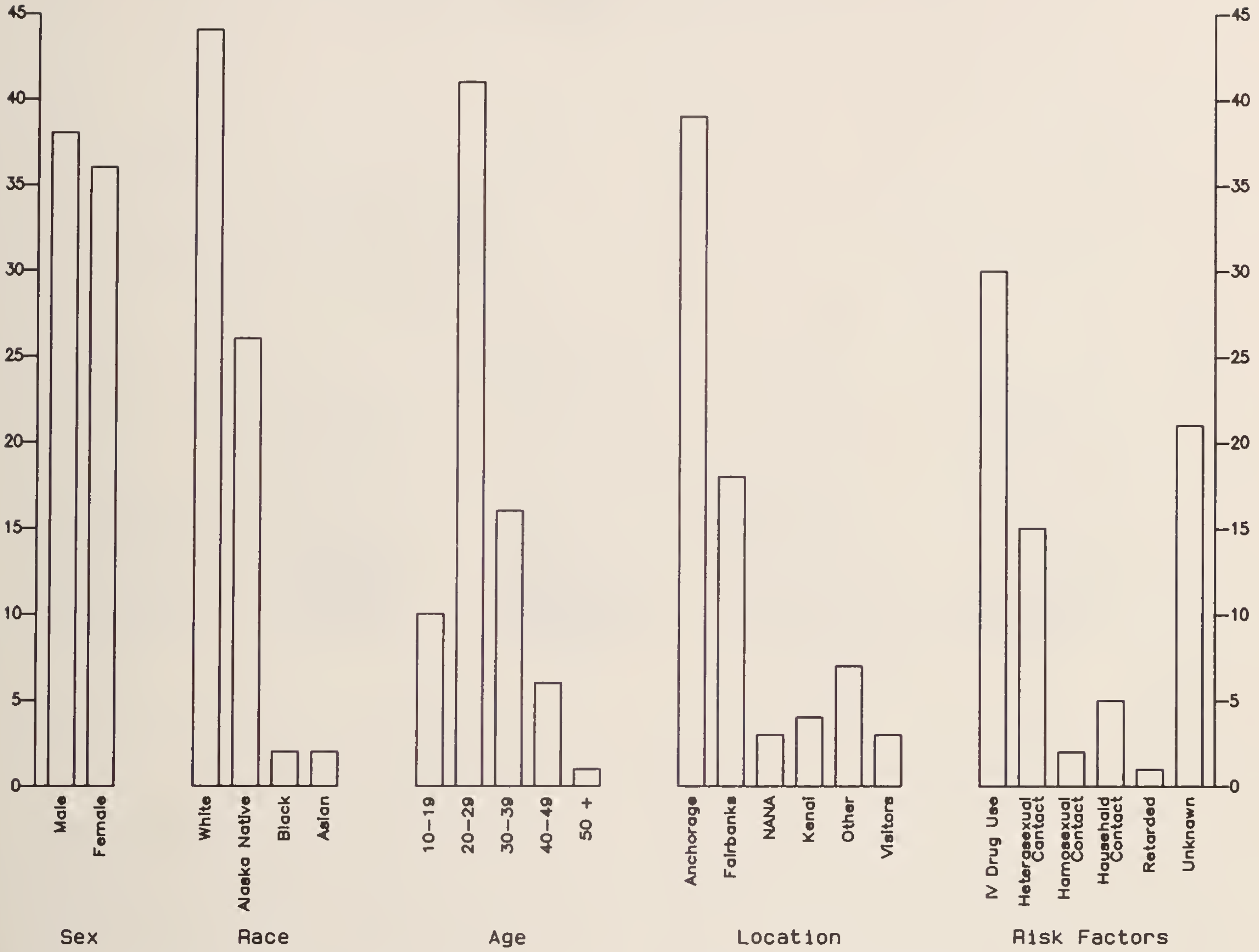
Communities in northern and southeastern Alaska showed little evidence of hepatitis B infection. For the past two years, most acute cases of hepatitis B have occurred in urban populations, particularly Anchorage and Fairbanks, and have been associated with intravenous drug use or sexual activity (Figure 2).

Hepatitis B in Alaska is not only an Alaskan Native problem.

Hepatitis B is a sexually transmitted disease that affects all races (11). The routes of hepatitis B transmission are similar to those of HTLV-III virus (12), and worldwide concern about the spread of AIDS should increase the general awareness of the hepatitis B danger as well. However even though hepatitis B is far more infectious than HTLV-III (13), people are less concerned because hepatitis B is not as frequently fatal. Both hepatitis B and HTLV-III infection are sexually transmitted diseases of special concern because they can subsequently be transmitted from infected pregnant women to their infants (14). Infants infected with hepatitis B virus at or soon after birth may not appear to be sick but are likely to become highly infectious, chronic carriers of the virus (15). As demonstrated repeatedly in Alaska and in many other parts of the world, these young infected children can spread the

Figure 2

Acute Cases Hepatitis B, Jan. – Sept., 1986 (N = 74)



infection to their siblings and playmates by sharing articles such as toothbrushes (16) and substances such as chewing gum (17).

A high prevalence of hepatitis B antigen and antibody has been documented in the sera of Alaskans of Asian origin (Figure 3). This finding is not surprising since there are an estimated 200 million carriers in the world today, primarily in Asia and sub-Saharan Africa (18). Although a comprehensive vaccination program for Asian children would be desirable, such an ambitious program cannot be undertaken at this time due to funding restrictions. Screening of pregnant Asian women is strongly recommended in order to recognize and treat prophylactically the infants at the greatest risk of becoming chronic carriers (19).

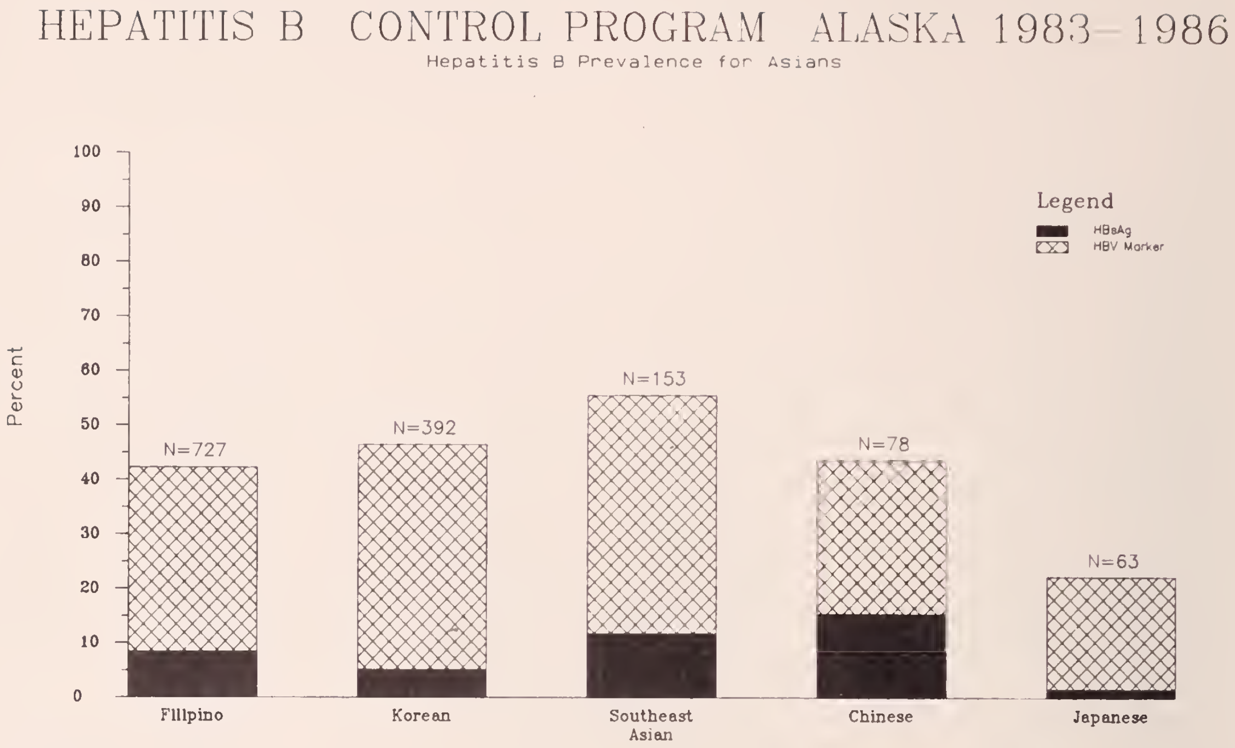
Hepatitis B carriers will continue to be present in the Alaska population for years to come.

Approximately 1400 chronic carriers were identified in the Native population as a result of the screening program and are under surveillance by the Alaska Native Health Service and AIL-CDC. Once established, the carrier state is slow to resolve, particularly in young children (15). Many carriers are children and teenagers just entering the age of sexual activity and childbearing. Since all Alaskan Native babies born since 1984 in the Yukon Kuskokwim and Bristol Bay regions have received hepatitis B vaccine, few additional children should become infected. Routine vaccination of all Alaskan Native babies is now recommended. Vaccine for Alaskan Natives is provided free of charge by the Alaska Native Health Service.

In addition to the Alaska Native carriers, 400 HBsAg positive individuals were identified, belonging primarily to identified high-risk groups such as Asians, homosexual males, and intravenous drug users (Figure 4). Screening of persons in these risk groups has not been comprehensive and will continue to present an ongoing challenge.

Health care workers in Alaska have been receptive to

Figure 3



the vaccination program; a large percentage of hospital employees, dental and medical office staff, and emergency medical technicians have been vaccinated. Between 1983 and 1986, the state provided hepatitis B vaccine free-of-charge to hospitals and medical and dental offices throughout Alaska for vaccination of employees with direct patient contact. A survey of Alaskan hospitals in 1986 indicated that an average of 50% of at-risk employees completed the vaccination series, and more were in progress.

Hepatitis B vaccine is safe.

Approximately 30,000 persons have received hepatitis B vaccine during the program. The Alaska Native Health Service and Regional Health Corporations are continuing to vaccinate many more. No significant adverse reactions to the vaccine have been reported — few people have even complained of sore arms. In Alaska, as elsewhere, there has been no evidence of HTLV III transmission related to hepatitis B vaccine administration (25).

Hepatitis B vaccine is effective.

Seroconversion, as documented by the presence of HBsAb on ELISA testing, has occurred in over 90% of the Alaska Natives included in a 3% sampling of those vaccinated by the Division of Public Health. Less optimal seroconversion has been reported in health care workers vaccinated in various parts of the United States with failure attributed in part to poor absorption of vaccine when injected into the fatty tissue of the buttocks rather than the arm (26).

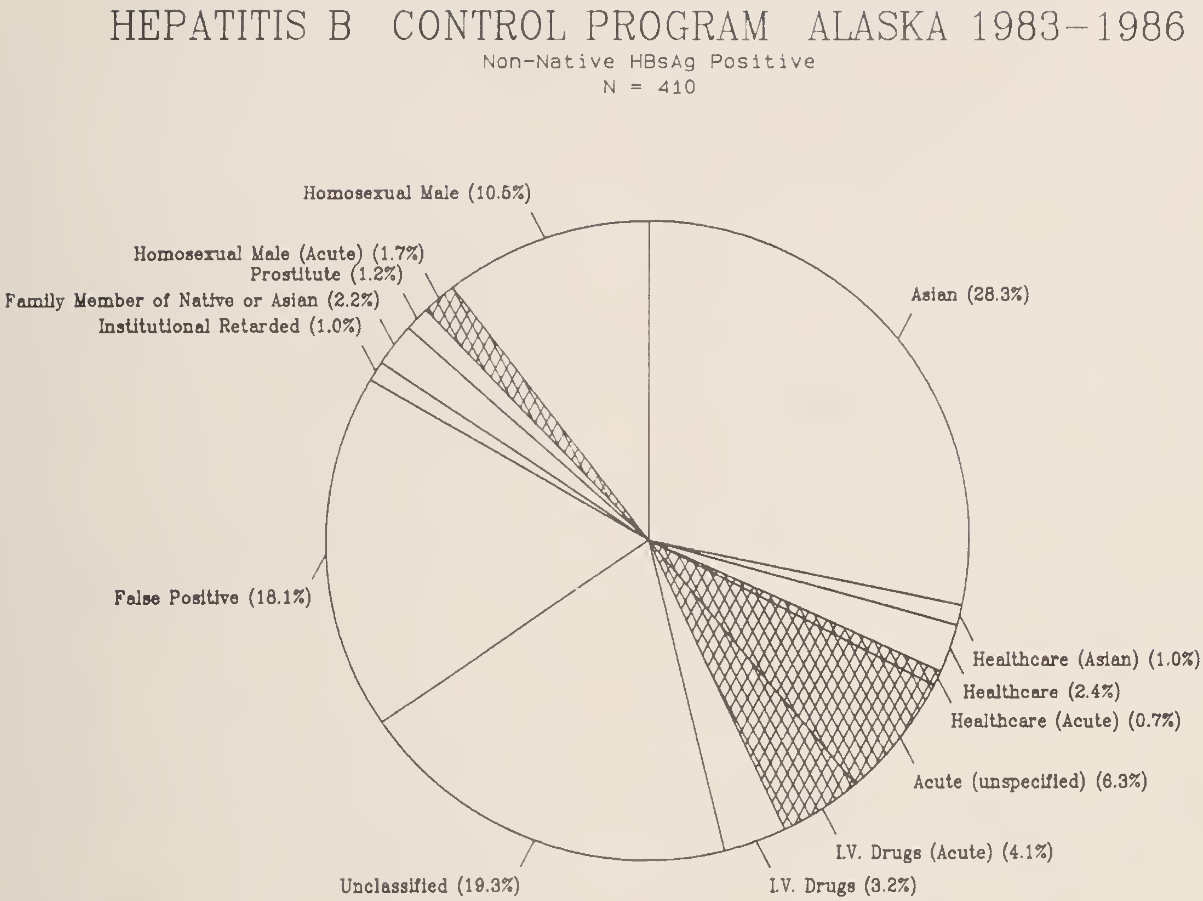
Controlled studies of seroconversion rates in Alaskan health care providers were not undertaken, because the administration of vaccine was delegated to the hospital personnel, and there was no consistent policy for post vaccination antibody testing. Several hospitals have elected to test for seroconversion, especially when injections were given in the buttocks. Results appear to be similar to those reported elsewhere. Revaccination has been effective in most instances, although seroconversion rates in Caucasian health care workers do not appear to be as favorable as those in the Alaskan Native population.

In AIL–CDC field trials, persons over 50 years of age were noted to have lower seroconversion rates than younger persons. Several of the documented vaccine failures in the State administered program occurred in elderly Natives.

A Comprehensive Vaccination Program can be Effective in Reducing Transmission of Hepatitis B Infection.

Acute hepatitis B is rarely seen now in the Yukon–Kuskokwim villages. The four cases reported in 1985 and 1986 from that region occurred in non–immunized Caucasian adults as a result of intimate contact with known carriers.

Figure 4



DISCUSSION

Eradication of Hepatitis B from the entire population of Alaska cannot be achieved in the short-term.

In the closely knit rural villages, it has been possible to identify all individuals susceptible to hepatitis B infection and to seek them out for vaccination. Elsewhere in Alaska those persons at the greatest risk are difficult to reach by usual public health measures (27). Potential drug users and homosexuals rarely identify themselves at a stage when vaccination would be indicated. Over 50% of the known male homosexuals and IV drug users who have been screened in Alaska show evidence of having already had hepatitis B, and very few of those still susceptible have elected to receive hepatitis B vaccine.

Comprehensive vaccination of all children might eventually eliminate hepatitis B infection but would not be feasible at this time with the high cost of the vaccine.

Prenatal screening with HBsAg as the only test may be misleading.

Prenatal screening of high risk individuals with only HBsAg fails to identify high risk families. Hepatitis B infected mothers may already be immune, but siblings may be carriers (20). A partial solution to this problem in Alaska was achieved by recommending that all Native and Asian newborns receive hepatitis B vaccine. An additional solution is also to screen prenatal, high risk individuals for HBeAb. If either test is positive, the family should be screened and susceptibles vaccinated.

False positive tests for HBsAg occur relatively frequently. Of 410 non-Native sera that initially tested positive for HBsAg by ELISA at the Northern Regional Laboratory, 74 (18.1%) subsequently proved on repeat testing to be false positives. False positive HBsAg results were a particular problem when low risk prenatal women were screened. The predictive value of a single HBsAg screening test in low prevalence populations is low—well documented by our experience in screening low risk pregnant women in the Matanuska Valley north of Anchorage.

The positive predictive value is only 10% for a single HBsAg test with 99% specificity in an area with a HBsAg prevalence as low as in the Matanuska Valley (0.4%) (21). Only one of ten prenatal women in the Matanuska Valley who initially had a positive test for HBsAg by ELISA showed markers confirming hepatitis B infection on repeat testing. The one woman whose positive markers were confirmed was initially positive for HBcAb in addition to HBsAg, while the other nine were negative for HBcAb.

Prenatal screening at the Northern Regional Laboratory includes testing for HBcAb in addition to HBsAg. Absence of HBcAb provides an early indication that the HBsAg test may be a false positive; repeat testing can allay concern and unnecessary administration of HBIG.

A positive HBsAg test without a positive HBcAb test can occur in early hepatitis B infection. But, in our experience, this finding has occurred in less than one percent of the instances where HBsAg was positive and HBcAb was negative. Some of these false positive HBsAg results were due to variations in the ELISA kits. Improved tests may be forthcoming.

False positives have also occurred when the testing was done by Radio-Immune Assay (RIA), evidenced by the fact that some Alaskan Natives originally placed on the Indian Health antigen positive registry as the result of RIA testing have subsequently been removed from the register after repeat testing.

The most meaningful serological tests are not always obtained on suspected cases of acute hepatitis.

Hepatitis A and hepatitis B have similar clinical manifestations, and the presence of hepatitis B infection should not be presumed because a person belongs to a recognized risk group. Serological tests for both hepatitis A and hepatitis B markers should be obtained from all patients presenting with acute disease. Hepatitis A tests should include HAV-IgM in addition to HAV-IgG because HAV-IgM is not always included in the routine hepatitis panel offered by private laboratories. A positive test for HAV-IgG would usually be the result of a previous infection (22). Testing for hepatitis B core-IgM should also be requested on suspected acute cases, because a positive core IgM test is often the only way to differentiate an acute case of hepatitis B from an unrelated intercurrent infection in a chronic hepatitis B carrier (23).

The Northern Regional Laboratory will perform these tests routinely, in additional to HBsAg, HBsAb and HBcAb on sera that are specifically designated as coming from symptomatic persons. Such sera is tested as a priority; results are telephoned directly to the submitter. Results can usually be obtained more rapidly than from out-of-state laboratories.

Testing for the antibody of delta hepatitis, a virulent form of hepatitis associated with hepatitis B infection, is also available at the Northern Regional Laboratory and is performed routinely in HBsAg-positive sera from severe acute cases or IV drug users (24). Although several Alaskan sera have shown delta antibody, as yet, no acute cases of delta hepatitis have been recognized.

Many cases of acute Hepatitis are not reported to Public Health Authorities although both Hepatitis A and Hepatitis B are reportable diseases.

The Division of Public Health Epidemiology Office has since 1984 directed efforts toward the follow-up of suspected cases of hepatitis. As a result of increased investigations and improved case-finding, the number of hepatitis B cases appears to have more than doubled in the first six months of 1986 compared to 1985. This increase is probably the result of the increased surveillance and case-finding—at least 29 of the 79 cases would not have been reported otherwise, and unreported 1985 cases have also been detected. Over 50% of the hepatitis B cases in both 1985 and 1986 were related to intravenous drug use, either directly, or through sexual contact with drug users.

FUTURE OF THE HEPATITIS B PROGRAM IN ALASKA

The substantial state and federal subsidization of hepatitis B control efforts in Alaska will soon be ending. State funding has already been severely curtailed, and the special allocation of Indian Health funds will end in 1987. Much has been accomplished towards preventing further spread of hepatitis B, but the danger is not over. The experience of the past four years should make it possible to allocate scarce remaining resources in the most cost-effective manner.

Screening Priorities

Further comprehensive screening is no longer indicated for confirming prevalence data in segments of the Alaskan population. Ample information has been accumulated regarding hepatitis B prevalence in various regions of Alaska. Most of the still susceptible Alaskan Natives in high prevalence areas have already been screened and vaccinated.

Screening, if linked directly to vaccination, should continue for high risk, non-Natives including:

- High-risk, pregnant women so that infants at risk are identified and can receive prophylactic treatment;
- Male homosexuals and IV drug users who make a commitment to receive all three doses of hepatitis B vaccine should they prove to be susceptible;
- Contacts, particularly sexual contacts, of acute cases should be screened prior to or along with the administration of HBIG and hepatitis B vaccine since they may be the source of the infection;
- Health care workers in Alaska have shown a low prevalence of hepatitis B infection unless they belong to other high risk groups. Prevaccination screening is not essential unless desired by the individual or the institution in which they are employed;

- High risk infants should not be screened prior to vaccination because screening results do not reliably indicate immunity. Children less than one year of age frequently show antibody temporarily as the result of passive transfer from their mothers.

Screening of high risk groups to identify carriers can be justified if counselling is provided to explain the carrier state and to emphasize precautions that may prevent the spread of infection. Persons recovering from acute hepatitis B infections should be rescreened in six months to see if they have seroconverted.

Alaskan Native carriers are being followed with alpha-fetoprotein determinations which may be helpful in detecting early resectible PHC, although the test is not yet licensed for that purpose. Prospective studies of Asian carriers currently underway in Japan, Taiwan, and cities like Los Angeles and Philadelphia may result in future recommendations for the medical follow-up of chronic hepatitis B carriers. A program has not as yet been developed in Alaska for surveillance of non-Native, HBsAg carriers.

Vaccination Priorities

The two goals of a hepatitis B vaccination effort are the prevention of acute cases and the prevention of the development of the carrier state. Dental professionals and health care workers who may come in contact with carriers in the course of their employment are at risk of acute infection and should be vaccinated for their own protection (29). Transmission of HBV from health care providers to their patients is rare but has occurred and continues to be a concern (30, 31). Vaccination of students prior to patient contact is recommended. Screening should not be necessary, since the prevalence of markers in health care students is low (32). A new hepatitis B vaccine made from yeast rather than human blood will be available commercially in 1987 and should be attractive to some health care workers who have not yet elected to receive hepatitis B vaccine (33).

Sexual contacts of acute cases and known carriers can benefit from preventive treatment if still susceptible (11). Whenever possible pre-exposure vaccination of potential sexual contacts of known carriers should be recommended. A woman exposed to hepatitis B at the time she conceives may develop the acute infection while in the third trimester of pregnancy, causing the infant to become infected (34).

Prevention of the carrier state in infants continues to be the highest priority in a hepatitis B control program. The Alaska Native Health Service plans to continue the policy of vaccinating all Native infants at or shortly after birth. The Division of Public Health also recommends vaccination of Asian newborns and children less than 10 years of age without screening, particularly in families with continued contact with relatives from the high prevalence countries.

Vaccination of susceptible homosexual males may also be of particular importance in controlling the spread of hepatitis B since homosexual carriers infected with HTLV-III have been observed to remain HBeAg positive for a prolonged period of time and are highly infectious. Failure to develop anti-HBe among HTLV-III/LAV infected persons may be a reflection of impaired T-cell immunity (39).

Vaccination of susceptible IV drug abusers is the only available means of preventing the spread of the deadly delta hepatitis since infection with hepatitis B is a prerequisite for delta infection (35). Preventing hepatitis B infection will have the added benefit of preventing the possibility of infection with the delta agent. Delta hepatitis, previously occurring in the United States mainly in east coast drug users, is now being seen with increased frequency among male homosexuals on the west coast (36).

No definite recommendations have been made as yet regarding the necessity for hepatitis B vaccine booster doses. HBsAb titers are known to decrease with time (37), but subsequent exposure to hepatitis B virus has resulted in an amnestic response in persons who initially responded to the vaccine.

CONCLUSIONS

1. Eradication of hepatitis B in Alaska is not feasible as long as chronic carriers are present in the population. Vaccination of all susceptible Alaskans is not cost-effective at this time. Transmission of the infection can be minimized by prompt case reporting and epidemiologic follow-up;
2. All physicians and nurses need to assess risk factors in each individual patient and to recommend the most appropriate screening, vaccination, or further diagnostic testing;
3. Health care employers should develop and implement policies and procedures in accordance with national recommendations to protect their patients and employees from hepatitis B infection;
4. The Division of Public Health will continue to provide:
    - serological testing for hepatitis A and hepatitis B upon physician request,
    - epidemiologic follow-up of acute cases,
    - provision of hepatitis B vaccine free-of-charge for high risk persons who can not obtain it otherwise.
5. *All* patients with suspected acute hepatitis should receive appropriate laboratory tests to make a definitive diagnosis. Diagnostic serologic tests are available free-of-charge through the Northern Regional Laboratory, Section of Laboratories, Division of Public Health. Results are available in

48-hours or less and can aid in decisions about use of immune serum globulin, HBIG, and hepatitis B vaccine;

6. All suspected or diagnosed cases of hepatitis should be reported promptly to the Epidemiology Office, Division of Public Health so that investigation can trace the source, identify individuals at risk, and facilitate prophylactic treatment;
7. All physicians and other health care providers at risk should get vaccinated.

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Prevalence of Drug-Taking Behavior Among Alaskan Youth: Comparisons With National Findings[1]

by Bernard Segal, Ph.D.[2]

ABSTRACT

The present research identified the prevalence and patterns of drug-taking behavior among a statewide sample of youth in grades 7-12. The findings indicated that lifetime experience with psychoactive substances was fairly high. When a comparison was made with national data, prevalence rates within Alaska were found to be quite higher than rates reported among youth in the lower-48 states. Some of the reasons for this high level of drug use and the implications of the findings are discussed.

INTRODUCTION

The United States has witnessed an increase in the frequency and intensity of substance abuse since the late 1960's. The non-medicinal use of illicit mood-altering drugs by elementary, junior and senior high school students, in particular, has become a problem of national concern. Although legal and social sanctions exist against non-medicinal use of psychoactive drugs for recreational or social purposes, drugs continue to be taken, and those youth who take them place themselves at risk for potential legal, social and health problems.

Interest in learning more fully about the nature and extent of drug-taking behavior among the nation's youth prompted the National Institute on Drug Abuse (NIDA) to sponsor a number of national surveys and specific research studies (1-3). Alaska, however, was not included in any of these projects. The current research was undertaken to gain comparable information about Alaska because educators, community planners, policy makers, legislators, and governmental officials and authorities needed to have contemporary knowledge about drug abuse in the state to most efficiently allocate resources to address the problem. High levels of drug use were anticipated due, in part, to the state's last frontier atmosphere which helped to attract many troubled people to the individualistic risk-taking attitude that prevails in the youthful population, and to significant disposable income, among other contributing factors.

The specific research objectives of this study were: (1) to assess the nature and extent of drug-taking behavior among Alaskan youth, (2) to examine age-cohort differences with respect to drug taking behavior, (3) to identify psychosocial correlates of drug use, and (4) to explore some of the implications that such phenomena have for treatment and prevention programs. This paper presents the major findings pertaining to the prevalence of drug use in Alaska among youth, compares the findings with results from national surveys, and briefly reviews their implications. The findings pertaining to psychosocial correlates and age differences, as well as a complete description of the project, have been presented elsewhere (4-6).

METHODS

From 1981 through 1982 an extensive statewide study was undertaken to estimate the prevalence of drug use among Alaskan youth. The statewide survey involved eight widely separated urban and rural school districts, representative of the different regions of Alaska except for the Aleutian chain. The locations were Anchorage, Barrow, Bethel, Fairbanks, Juneau, Kotzebue, Nome, and Sitka. Sites were selected in order to obtain a representative sample of the state's junior and senior high school students, rather than sampling school districts. The schools in the sample contain over 75% of the state's junior and senior high school students. Representative samples in the Anchorage and Fairbanks school districts were obtained from each of the junior and senior high schools. Districts in the other locations contained only one junior and senior high school, of which the entire school populations were surveyed.

The sampling procedure within the schools involved two methods, each contingent upon the conditions prescribed by the school district: (1) a random sample from among all students in grades 7-12 whose parents

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gave parental consent (Anchorage, Fairbanks and Juneau); and (2) surveying the entire school population of students in grades 7–12 (the remaining five locations).

The first procedure could have presented a problem if the response rate for parental disapproval was high. The opposite happened. Very few (less than 2%) parents objected, thereby allowing a sufficiently large school population from which to obtain a representative sample. The requirement of parental permission did not appear to result in an underrepresentation of drug using students. This became evident when the results were analyzed separately by method of collection, by district, and by region. Despite the different sampling procedures the results were strikingly uniform across the state (6); no significant differences were noted, and the data was aggregated to form a comprehensive data base. The results reported herein are from the aggregated data.

Although the content of each survey was practically identical, in some cases wording and format were simplified to facilitate translation or to comply with school district's regulations. Survey administration was conducted or supervised by a member of the research team during a given school day. A total of 3,609 completed student surveys were obtained (which represented a 99% response rate); males and females were equally distributed (51% males, 49% females), and ages ranged from 11 to 19. The survey questioned nonprescriptive use of marijuana, stimulants, cocaine, hallucinogens, depressants, heroin, inhalants and tranquilizers. Inquiries were also made about use of alcohol and tobacco. Additional questions concerned age of first use of each drug, frequency and recency of use, reasons for use, and other related items.

RESULTS AND DISCUSSION

1. Lifetime Experience

Table 1 contains figures pertaining to opportunity to try different mood altering substances and the percentage of youth who reported having experienced one or more of these substances. Except for hallucinogens (48.1%) and heroin (29.9%), over half those students who had a chance to try one of the other drugs did so: marijuana 74.8%, depressants 71.2%, tranquilizers 72.6%, stimulants 76.2%, and cocaine 63.3%. It is readily apparent that large numbers of students have indicated an opportunity to try different types of psychoactive chemicals and that a correspondingly high number seem to have taken advantage of such opportunities.

Consistent with current national trends (1–2), marijuana is the drug that most students have had a chance to try and do try; half of the students (49.5%) reported using marijuana one or more times. It thus appears that marijuana was available around the state, and that it was the most widely used illicit substance. In contrast to this finding, and also consistent with national trends, heroin was the drug students were least exposed to and the one least used (2.2% reported having tried this drug). Whether this finding was attributable to an unavailability of heroin in the state or to a reluctance by students to try it is difficult to discern. It may be that both conditions contributed to the low reported incidence of experience.

Table 1

Opportunity to Try and Trying Psychoactive Drugs Lifetime Experience

(N = 3,609)

Drug	Percent of Sample Having Opportunity	Percent Having Opportunity and Trying	Percent of Total Sample Trying
Marijuana	66.1	74.8	49.5
Hallucinogens	18.1	48.1	8.7
Cocaine	29.0	63.3	18.3
Heroin	7.2	29.9	2.2
Inhalants	26.8	61.5	16.5
Stimulants	35.7	76.2	27.2
Depressants	20.1	71.2	14.3
Tranquilizers	15.9	72.6	11.5

Stimulant-type drugs are second to marijuana in terms of lifetime experience (27.2%), followed by experiences with cocaine (18.3%), inhalants (16.5%), depressants (14.3%), tranquilizers (11.5%), and hallucinogens (8.7%).

The pattern of drug experience reported by the students suggested that they tend to have avoided the more "addictive" substances, such as heroin, and others that can induce severe adverse effects (e.g., inhalants, depressants and tranquilizers). What seemed to be preferred, instead, was the use of drugs that induce more pleasant effects such as a state of euphoria (marijuana) or a "rush" as is associated with stimulants or cocaine.

Of those who tried drugs, the largest percentage (35.9%) used only one drug, 21.3% tried two drugs, 11.5% experienced three, 29.8% tried four or more drugs, and 1.5% tried all eight of the drugs listed in the questionnaire.

2. Frequency and Recency of Drug-Taking Behaviors

The survey inquired about the frequency and recency of students' drug-taking behaviors. One set of these questions (concerning frequency of use) differed for the Anchorage sample, thereby precluding their inclusion in the report of aggregated data. The results of the findings

Table 2

FREQUENCY AND RECENCY OF DRUG-TAKING BEHAVIOR
Past Year Experience

Percent of Students Who Have Tried/Taken a Drug

	(N = 3,609)		Frequency* (N = 2,021)						
Drug	Percent of Sample Responding	Percent of Sample Who Tried Within Past Year	Once a Month or Less	2-3 Times a Month	Once a Week	2-5 Times a Week	Daily	More Than Once a Day	Total Once or More
Marijuana	88.0	42.6	19.0	7.1	4.0	6.0	2.6	2.8	41.5
Hallucinogens	82.0	7.1	4.9	0.7	0.2	0.2	0.2	0.0	6.4
Cocaine	83.0	15.6	11.0	2.5	0.6	0.7	0.3	0.4	15.5
Heroin	81.9	1.7	1.7	0.2	0.1	0.1	0.1	0.3	2.5
Inhalants	82.6	11.9	7.4	1.4	0.5	0.4	0.4	0.4	10.5
Stimulants	83.1	22.0	12.2	4.0	1.7	1.3	0.8	0.9	20.9
Depressants	82.2	11.8	7.3	2.1	0.6	0.6	0.2	0.5	11.3
Tranquilizers	81.7	11.4	6.5	1.4	0.6	0.3	0.2	0.4	9.4

*Because of missing responses, those who report having tried a drug in the past year will not always correspond to the percent who reported ever trying a drug.

describing frequency of students' use thus represent an aggregation of only seven districts (excluding Anchorage), while the findings pertaining to the recency of use represent the total sample.

Table 2 describes the recency and frequency of drug-taking behavior during the past year. The results indicate that marijuana is the drug experienced most, with 41.5% of the students indicating having experienced it one or more times, and 42.6% having used it during the past year.

Experiences with stimulants and cocaine are less extensive than those shown for marijuana, but are, respectively, the most commonly used substances. Most of the students who tried them did so less than 10 times and once-a-week or less. Experiences with the other drugs tended to be less frequent, with students expressing modest experiences with depressants, inhalants, and tranquilizer type substances. Except for marijuana, and to a lesser degree stimulants and cocaine, use of other drugs appeared to have been chiefly experimental.

3. Gender and Drug-Taking Behavior

Table 3 indicates that a higher percentage of males experienced all the mood-altering drugs except stimulants, for which females showed a slightly higher percentage. This finding is comparable to that reported by Johnston et al. (2) in describing drug use among high school seniors.

Table 3

LIFETIME EXPERIENCES WITH PSYCHOACTIVE DRUGS BY GENDER

Females and Males Who Reported *Ever Having Tried* a Drug*

	MALES (N = 1,770)	FEMALES (N = 1,732)
Drugs	Percent of Males who Tried a Drug	Percent of Females who Tried a Drug
Marijuana	51.1	48.3
Hallucinogens	10.5	8.7
Cocaine	19.8	17.0
Heroin	3.2	1.2
Inhalants	17.9	15.1
Stimulants	26.3	28.2
Depressants	15.4	13.6
Tranquilizers	12.4	10.9

*107 students did not report gender.

4. Drug-Taking Behavior by Grade

As reflected in Table 4, there was a steady increase in experiences with all types of drugs rising to a peak at grades 11 and 12. The major exception to this pattern was for inhalants, where incidence of use peaked sharply

Table 4

DRUG-TAKING BEHAVIOR BY GRADE

Percent Within Each Grade Who Reported Trying/Taking a Drug

Students Grade 7-12

	Grade*					
	7	8	9	10	11	12
Drug	(N = 665)	(N = 685)	(N = 603)	(N = 658)	(N = 564)	(N = 345)
Marijuana	23.3	38.7	49.1	60.6	69.9	69.6
Hallucinogens	2.9	5.5	8.0	10.9	13.8	15.4
Cocaine	4.8	12.1	13.9	21.9	31.6	37.1
Heroin	0.9	1.8	2.3	2.6	4.4	1.2
Inhalants	12.0	18.0	14.4	18.8	18.8	17.4
Stimulants	8.1	20.0	25.2	36.0	42.6	40.9
Depressants	6.2	12.4	13.4	18.7	21.1	18.0
Tranquilizers	3.8	9.6	10.0	15.7	18.3	14.5

*304 students did not report grade levels.

in the eighth grade. It may be that inhalant substances (such as gasoline, paint thinner, sprays, etc.,) are more readily obtainable and less expensive for these youngsters than other substances, allowing emulation of the "highs" that older students achieve through the use of the "hard" drugs.

It may be inferred from this table that an orderly progression of drug use may exist. Segal (4), when analyzing the relationship between age and initiation into drug-taking behavior, did find a predictable sequential pattern of drug use, beginning with marijuana, followed by stimulants, cocaine, depressants, tranquilizers, and hallucinogens.

The data in Table 4 also permit a general comparison of drug use between junior (grades 7–9) and senior high school (grades 10–12) students. The findings indicate clearly that more senior high students have tried drugs than junior high students. (A separate Chi Square analysis comparing drug use between the two groups of students found that the differences are statistically significant.)

Although the drug-taking behavior was more extensive among high school students, the pattern of use was similar to that of junior high school students. Marijuana, stimulants and cocaine were the three most experienced drugs, respectively, while heroin, tranquilizers and hallucinogens were among the least experienced. Moderate use of inhalants and depressants was noted.

5. Comparison with National Survey Data

Table 5 provides a comparison of the Alaska data with findings from the 1982 National Survey on Drug Abuse (1). It should be noted that this comparison is not based on similar methodologies. The Alaskan study is based on responses to questionnaires, the national survey is based on responses to personal interviews; also the ecological environments of the two samples differ greatly. The comparison is provided, however, in order to demonstrate how patterns of use among Alaskan youth generally compare to those reported for youth in the "lower-48" states.

Table 5

COMPARISONS OF ALASKA WITH NATIONAL SURVEY FINDINGS OF LIFETIME EXPERIENCES WITH PSYCHOACTIVE SUBSTANCES

Drugs	**Alaska Youth 12-17 Years (N = 3,103)**	**1982* National Survey 12-17 Years (N = 1,581)**
Marijuana	47.4	26.7
Hallucinogens	7.9	5.2
Cocaine	16.6	6.5
Heroin	2.3	<0.1
Stimulants	25.9	6.7
Depressants	14.0	5.8
Tranquilizers	11.1	4.9
Alcohol	71.7	65.2
Tobacco	55.0	49.5

*Miller and Cisin (1).

Table 6

COMPARISONS OF LIFETIME EXPERIENCE WITH PSYCHOACTIVE DRUGS BY HIGH SCHOOL SENIORS

	Percent Trying			
Drug	Alaskan Seniors (N = 345)	1982* National Seniors (N = 17,500)	1983* National Seniors (N = 16,300)	1984* National Seniors (N = 15,900)
Marijuana	70**	58	57	54.9
Hallucinogens	15	13	12	10.7
Cocaine	37	16	16	16.1
Heroin	1	1	1	1.3
Inhalants	17	13	14	14.4
Stimulants	41	36	36	27.9
Depressants	18	12.8	13.6	13.3
Tranquilizers	15	14	13	12.4

*Source: Johnson (2), National Institute on Drug Abuse.
**Figures are rounded to nearest whole number.

The comparisons show that the rates for experience with drugs among Alaskans aged 12 17 exceeded the rates obtained in the national sample for every substance. The magnitude of the differences range from a low of 2:1 to a high of 3:1. It is apparent that drug-taking behavior occurred at a higher rate among Alaskan youth than among those reported in the national survey, but because of differences in sampling procedures and time of sampling, these differences should be interpreted as a relative comparison rather than exact differences. Nevertheless, the question that arises is: Why are the differences so extensive? One answer may lie in the sampling procedure; youth may be less reluctant to report drug use in a questionnaire than in a personal interview. Another possible explanation is that drug use in Alaska is actually higher. The latter explanation tends to be supported when a comparison is made between high school seniors in Alaska and seniors in Johnston et al's (2) national study. Representative findings from these more comparable surveys are presented in Table 6. Except for heroin, drug experience rates for Alaskan seniors exceed the rates obtained in the national senior sample.

CONCLUSIONS

The results of this study indicate a pattern of drug-taking behavior that is consistent among adolescents across the state. Furthermore, prevalence levels for lifetime experiences in Alaska, despite limitations in comparing the Alaskan data with national household data, are higher than the prevalence rates reported in the national survey, and in the national survey of high school seniors. Another possible limitation to the study is the fact that it is not known whether the respondents who reported taking a drug actually took the "real thing" as opposed to a "look alike" or substitute substance, except for marijuana.

While this problem is common to all forms of survey research, it need not detract from the basic findings. What is important to consider is that the respondents, regardless of whether they may have taken real or substitute drugs, reported that they were involved in some form of drug taking behavior, and it is *their* account of their behavior in which we are interested. Additionally, the issue of the reliability and validity of self reports concerning drug taking behaviors always comes into question, but sufficient research has been conducted on this problem to conclude that self reports are generally reliable and valid and tend to be free from systematic bias.

The major question that arises is: Why are the prevalence rates so high in Alaska? Part of the answer seems to be that drugs are readily available, and that many young people are actively interested in seeking these drugs. Furthermore, mood altering drugs may have become incorporated into the "Alaskan life style." In this context, experimental or recreational drug taking behavior is viewed primarily as a social phenomenon and not as deviant behavior. In Alaska this life style pattern takes on a special quality. Perhaps many who try or use drugs recreationally perceive their drug-taking behavior as part of a life style that represents a breaking away from the traditional establishment—one that emphasizes a desire to develop free from conformity and a need to seek new, different, stimulating or exciting experience (5). Such behavior appears to be consistent with the "last frontier" individualistic, risk-taking behaviors practiced by many people in the state. Adolescents may tend to emulate these motives.

An important implication of this finding is that of determining more precisely how attitudes, life style or value systems espoused within the larger context of society exert an influence on the younger members of that society. For many of them, experimentation with drugs may not only serve to satisfy curiosity, but also may be a means of obtaining new and exciting experiences. While these motives may also prevail among "lower 48" youth (8), Alaska's unique environment tends to heighten their reinforcement. While all drug taking behavior cannot be accounted for by these reasons, research (5) has shown a strong relationship between these motives and such behavior.

The findings from the present study have significant implications concerning the prevention of drug-taking behavior. Different prevention goals may need to be devised for various points in the prevalence curves pertaining to first experiences with drugs. Since recent research (4, 9) has suggested that there may be critical periods for substance use initiation, there is every reason to underscore the importance of understanding these early use patterns as the first step in the design and implementation of primary prevention programs. The most logical strategy is to focus prevention efforts at the ages at which initiation into drug-taking behavior occurs. Prior to these ages prevention efforts should be directed at delaying or preventing the onset of non-medicinal use of mood-altering drugs; subsequent to these critical periods efforts should concentrate on the reduction of prevalence.

An important element of any prevention program should be a basic assumption that drug experimentation or limited recreational drug use may not necessarily represent deviant or pathological behavior. Rather, such behavior may have become a social norm, and youth in our society may be reflecting a form of behavior that has become acceptable among large segments of our society, even though it is illegal and poses a health risk. Prevention programs based on this premise should have a better chance at succeeding than programs that do not take this phenomenon into consideration. Prevention specialists, therefore, are faced with the task of developing a strategy that comes into effect after a drug has been tried—one that focuses on reducing use after initiation to or experimentation with mood-altering drugs has occurred.

In summary, it is evident that the lifetime prevalence rates for drug use were remarkably high in Alaska. It is also apparent that there is a need for a greater understanding of the factors that contribute to Alaska's high level of drug-taking behavior. The understanding of these factors should be applicable not only to Alaska but also to other areas of the country experiencing a rise in drug use among youth.

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Death in Anchorage in 1985

by Rodman Wilson, M.D.[1]

ABSTRACT

In 1985, 794 persons died in Anchorage. The age-adjusted death rate of 9.32 per 1,000 persons was 9% higher than the preliminary rate for the entire nation (8.57 per 1,000 persons) in 1984. Of individuals dying in Anchorage in 1985, approximately 28% died of cardiovascular disease, 25% of malignant neoplasm, 25% from violence broadly defined, and 23% from other conditions. The proportion dying violently in Anchorage was three times that in the nation in 1984. This may be because people are younger here or because life patterns are conducive to violence. Death certificates, although imperfect documents, are of great importance in understanding our society.

INTRODUCTION

Collection and analysis of vital statistics in Alaska is a function of state government (1). It is done by the Registrar, Bureau of Vital Statistics, Division of Planning, Policy and Program Evaluation, Department of Health and Social Services, State of Alaska.

Certificates of death, birth, fetal death, marriage, divorce, and the like, are sent for analysis to Juneau from 37 recording districts across the state. Selected data are transmitted from the Juneau office to the National Center for Health Statistics for incorporation into national enumerations.

In 1984, state law was changed to allow local health officers to examine vital documents. Anchorage is the only Alaskan community that has its own health officials and health department. I have perused the death certificates of all persons dying within the boundaries of the Municipality of Anchorage (co-terminal with the Greater Anchorage Area Borough) since September 1, 1984.

This is a presentation of deaths in 1985. Emphasis is upon violent deaths because they were so common in Anchorage in 1985 and on certain aspects of completing the physician's portion of death certificates.

METHODS

Underlying cause of death was identified on each certificate. An attempt was made in every instance to assign a proximate, and usually anatomic, cause of death rather than to accept a mechanism of death, such as cardiac arrhythmia, respiratory failure, inanition, or uremia as the "cause" of death. If an underlying cause of death was not apparent on a certificate, or if arcane abbreviations were used, I spoke to the physician who had signed the document or to the pathologist who had examined the body. This invariably confirmed or clarified the cause of death (insofar as the death was understood) and frequently added important information—such as autopsy findings not at hand when the certificate was filed, or time intervals between a surgical operation and death or between a stroke and a later infection.

It should be noted that these "edited" 1985 death certificates did not flow to the Registrar of Vital Statistics in Juneau for engrossment into Alaska and national statistics. Data in this study will not be the same as data for Anchorage that will eventually be published by the state office. My refined certificates, however, form a better base for analysis than the documents as received initially. Editing, however, was irregular in that I did not call every physician and was certainly not random. Physicians were spoken to in connection with 112 (14.1%) of 794 death certificates. This one-seventh represented the most troublesome certificates.

Disease specific rates were not calculated. The overall death rate for Anchorage was computed by using an Anchorage population estimate for mid-1985. Chi-square test was used to gauge significance of the differences between proportions of deaths among residents and non-residents, and Native Americans and others.

Violent deaths were those subsumed under the rubric "external causes of injury and poisoning" (E800-E999) of the International Classification of Disease, 9th Revision (ICD-9-CM) and deaths due to alcoholism (291, 303, 305, 425, 571, 577, 991).

RESULTS

In 1985, 794 persons died in Anchorage. Based on a mid-year, estimated population of 230,434, the crude death rate for Anchorage was 3.45 deaths per 1,000 persons. This rate is much lower than the crude death rate in the United States of 8.57 per 1,000 persons in 1983, the latest year for which figures are available (Table 1).

One might conclude that Anchorage is a healthful place to live. But when one corrects for the fact that our

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[1] Department of Health and Human Services, Municipality of Anchorage.

Table 1

1985 Crude Death Rate (CDR) for Anchorage Compared with Preliminary 1983 CDR for USA

	Number of Deaths	Mid-Year Population	CDR per 1,000
Anchorage	794 (1)	230,434 (2)	3.45
USA	2,010,000 (3)	234,537,000 (4)	8.57

1. Department of Health & Human Services, Municipality of Anchorage
2. Department of Community Planning, Municipality of Anchorage
3. National Center for Health Statistics
4. U.S. Department of Commerce, Bureau of Census

population is younger than that of the nation, the death rate in Anchorage rises to 9.32 (Table 2). The death rate in Anchorage was higher than the national death rate at every age group except in the 0–1 age category. The overall "age-adjusted" rate was 1.09 times—9% higher—than the crude death rate of the United States as a whole for 1983. Anchorage, thus, is not as salutary as it first appears.

Table 2

Age-Adjusted Death Rate (AADR) for Anchorage in 1985 Compared to Preliminary 1983 Crude Death Rate (CDR) for USA

	Rate per 1,000	Ratio
Anchorage, AADR*	9.32	1.09
United States, CDR	8.57	1.00

*Calculated by multiplying Anchorage age-group specific death rates by population in each age group in USA in 1983 to obtain total expected USA deaths if Anchorage experience had applied to the nation.

However, one should discount the rate of 9.32 to some extent because 203 (25.6%) of the 794 persons who succumbed in Anchorage resided elsewhere (Table 3). Anchorage is a referral medical center for large portions of the state, particularly for Alaskan Natives (Eskimo, Indian, and Aleut). Many persons came to Anchorage seriously ill or mortally wounded. If they died here, deaths were counted as occurring in Anchorage. By the same token, many Anchorage citizens died away from home, either recreationally, such as in a crash of a small aircraft, or in another state where they were visiting or to which they had gone for medical care. At least 51 such out-of-town deaths are known.* These fatalities, however, did not offset the large number of non-residents who came here and died. Thus, in calculating death rates, the population denominator for all deaths in Anchorage and the numerator for all Anchorage residents dying are suspect. These are common problems in analysis of vital events (2).

In Anchorage in 1985, approximately one quarter (27.6%) of 794 individuals died of cardiovascular disease, one quarter (24.9%) of cancer, one quarter (24.9%) from violence, and one-quarter (22.6%) of other conditions, including deaths in early life, infections, lung disorders, neurologic disease, and miscellaneous conditions (Table 4). Nationally in 1984, these proportions were, respectively, 30.9%, 22.1%, 8.3%, and 38.7%. Note that violent deaths were only one third as common in the nation as in Anchorage.

Violence, as defined here, included deaths from motor vehicles, aircraft, and bicycle accidents, fires, falls, blows, drowning, hypothermia, avalanche, drug or chemical overdose, suicide, homicide, and alcoholism including alcoholic liver disease. The National Center for Health Statistics does not include drug overdose, suicide, homicide, and alcoholism in its category of unintentional deaths designated "accidents and adverse effects" (E 800–E 949), but these other components of my definition of violence can be culled from national summaries to make up the national proportion of violent deaths mentioned above.

Persons residing within or outside Anchorage died in Anchorage of approximately the same disorders and in

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*Death certificates of 51 Anchorage citizens dying outside Anchorage have been recovered. Others have not yet been located. Eighteen (18) Anchorage residents died in the State of Washington, 7 in California, 4 in Arizona, 4 elsewhere in Alaska, 3 in Texas, 2 in Yukon Territory of Canada, 2 in Minnesota, 2 in Kansas, and 1 in each of 9 other states. Fourteen (28%) of these 51 individuals died of cardiovascular disease, 13 (26%) of cancer, 11 (22%) from violence, 6 (12%) from conditions mortal before age 1, and 7 (14%) of miscellaneous or unstated conditions. These proportions are approximately the same as among persons dying in Anchorage (Table 4) except that a higher proportion of infants died "Outside".

Table 3

Place of Residence of 794 Persons Dying in Anchorage in 1985

Anchorage				591	(74.4%)
Elsewhere in Alaska				164	(20.7%)
Wasilla	19	St. Paul	3		
Fairbanks	11	Valdez	3		
Kenai	11	Akiachak	2		
Palmer	9	Anchor Point	2		
Kotzebue	6	Barrow	2		
Chevak	5	Emmonak	2		
Seward	5	Kotlik	2		
Soldotna	5	Marshall	2		
Bethel	4	Nikiski	2		
Kodiak	4	Nome	2		
Big Lake	3	Port Lions	2		
Copper Center	3	Sand Point	2		
Delta Junction	3	Togiak	2		
Glenallen	3	Unalaska	2		
Homer	3	Yakutat	2		
Mountain Village	3	Other town, 1 ea	35		
Outside Alaska				39	(4.9%)
Washington	9	Minnesota	2		
California	5	Oklahoma	2		
Oregon	4	Other state, 1 ea	11		
Connecticut	2	Japan	1		
Hawaii	2	West Germany	1		
		Total		794	(100.0%)

similar proportions within broad categories of disease (Table 5) except that non-resident infants below age one died in significantly greater proportion than resident infants. This was probably because many very ill rural infants were brought to a newborn intensive-care unit in Anchorage.

Native Americans were victims of violent deaths even more commonly than Caucasians, Blacks, and Asians taken together. Of 133 Native Americans among the 794 persons dying in Anchorage in 1985, 44 (33.1%) died of accidents, alcoholism, and suicide, whereas 154 (23.2%) of 661 non-Natives died violently. The difference is significant ($p < .02$). A Native was not murdered in Anchorage in 1985. The high number of cases of alcoholism, suicide, and pedestrian accidents among Natives in Alaska is well-known (3,4).

When deaths in the United States in 1984 are compared to deaths in Anchorage in 1985 (separating violent deaths back into categories used nationally), the five leading causes in the United States—heart disease, malignant neoplasm, cerebrovascular disease, accidents, and chronic obstructive lung diseases—are also the leading causes of mortality in Anchorage, but rank order differs among the first five (Table 6). Violence and alcoholism are more prominent as causes of death in middle rankings (ranks 6–10) in Anchorage than in the United States. Diabetes mellitus, renal disorders, and septicemia were not among the first fifteen causes of death in Anchorage in 1985 in contrast to national figures for 1984. Sudden infant death syndrome (SIDS) ranked thirteenth in Anchorage, whereas it was unranked in the United States. Because numbers of deaths in categories after the first five are small in Anchorage, rankings could easily change from year-to-year without necessarily signifying important changes in patterns of death.

COMMENT

One should be cautious in interpreting and using information from death certificates because of their well-known imperfections (5–7). Caution is also in order here because analysis of only one year has been presented. It is obviously not possible to detect trends based upon only one year's analysis, and it may not even be possible to detect unusual clusters of deaths.

Nonetheless, one cannot help being impressed by the large number of violent deaths in Anchorage, particularly those due to motor vehicle collisions, vehicle-pedestrian encounters, homicides, suicides, unintentional drug overdose (frequently involving cocaine), and deaths from alcoholism.

Table 4

Categories of Death among 794 Persons Dying in Anchorage in 1985

	Number of Deaths	Percent of Deaths
Cardiovascular disease	219	27.6
Cancer	198	24.9
Violence	198	24.9
Death before age 1	51	6.4
Infection	45	5.7
Lung disease	38	4.8
Neurologic disease	17	2.1
Other cause	28	3.5
	794	99.9*

*Rounding artifact

One also notices in this survey how many deaths were "unnecessary" or preventable. One death from cancer of the lung occurred, on average, in Anchorage every week. Tobacco caused most of these, not to mention many of the deaths from cancer of the oral cavity, larynx, esophagus, and perhaps even the stomach, pancreas, kidney, bladder, and cervix. Smoking undoubtedly caused most of the 33 deaths from chronic obstructive pulmonary disease, and it certainly contributed heavily to cardiovascular mortality. Many Anchorage physicians now record tobacco use as the underlying cause of death in tobacco-related diseases, just as one identifies asbestos, arsenic, or an axe bifurcating a skull as the agent producing a disorder that leads to death.

Physicians often recorded a pathophysiological mechanism of death, such as respiratory, cardiac, hepatic, renal, or brain-stem failure, sometimes without mentioning an underlying disorder, such as, for example, chronic obstructive pulmonary disease, coronary atherosclerosis, alcoholic cirrhosis, chronic glomerulonephritis, cerebral hemorrhage, an underlying cancer, or extreme prematurity of an infant.

Fault lies to some extent with the death certificate itself. It asks for the "immediate cause", then "due to", a second "due to", and finally "other significant conditions". "Immediate" is intended to elicit the *proximate* cause of death, such as acute myocardial infarction, carcinoma of the lung, or Guillian-Barre' syndrome, and not necessarily what happened to the deceased in the last few moments of life. It is, however, entirely appropriate to record a sequence of events; for example, pulmonary embolism, due to crushed pelvis, due to motor vehicle accident.

Physiologic mechanisms of death are of interest but are less useful for public health purposes than anatomic causes of mortality because expressions of organ failure are relatively few, and they fail to differentiate the hundreds of disorders that can end, say, in respiratory failure.

"Alcohol" and "alcoholism" remain largely taboo as words to use on a death certificate unless the victim is a derelict. Such delicacy is understandable but difficult to excuse in a person trained in science. Deaths due to alcoholism, however, are often not difficult to spot, for they masquerade as "Laennec's cirrhosis", "chronic liver disease", "acute pancreatitis", or "chronic encephalopathy". And, of course, alcohol underlies many suicides, homicides, and unintentional violent deaths. More candor on the part of physicians would help delineate the dimension of alcoholism in Anchorage and Alaska. This, in turn, might lead to more effective ways to limit its ravages.

Violent deaths are more common in Anchorage than in the nation mainly because we are young. The young take risks and have less control than older persons. But youth alone does not explain the excess mortality from violence in Anchorage. There may be features of life here that condone or encourage conditions and behavior that lead to injury, alcoholism, drug use, depression, suicide, and homicide. These are questions for others to explore. Vital statistics, however imperfect, are the starting point for those who seek to understand disease, behavior, and culture.

Table 5

Number and Percentage of Deaths by Category of Disease Among 592 Residents and 202 Non-residents Dying in Anchorage in 1985

Disease Category	Residents (%)	Non-Residents (%)	Total (%)	Significance* Test
Cardiovascular disease	164 (27.7%)	55 (27.1%)	219 (27.6%)	NS
Cancer	154 (26.1%)	44 (21.7%)	198 (24.9%)	NS
Violence and adverse effects	145 (24.5%)	53 (26.1%)	198 (24.9%)	NS
Death before age 1	31 (5.2%)	20 (9.9%)	51 (6.4%)	$p = 0.025$
Infection	30 (5.1%)	15 (7.4%)	45 (5.7%)	NS
Lung disease	30 (5.1%)	8 (3.9%)	38 (4.8%)	NS
Other	37 (6.3%)	8 (3.9%)	45 (5.7%)	NS
	591 (100.0%)	203 (100.0%)	794 (100.0%)	

*Chi-square: NS = $p \geq 0.05$

Table 6

Leading Causes of Death in United States in 1984 and Anchorage in 1985

	Rank	
Cause	United States	Anchorage
Diseases of heart	1	2
Malignant neoplasms	2	1
Cerebrovascular diseases	3	4
Accidents and adverse effects	4	3
Chronic obstructive lung diseases and allied conditions	5	5
Pneumonia and influenza	6	11
Diabetes mellitus	7	
Suicide	8	6
Chronic liver disease and cirrhosis	9	7
Atherosclerosis	10	12
Nephritis, nephrotic syndrome, and nephrosis	11	
Homicide and legal intervention	12	8
Certain conditions originating in perinatal period	13	10
Septicemia	14	
Congenital anomalies	15	9
Sudden infant death syndrome		13
Senile and presenile degeneration of brain		14
Pulmonary embolism		15

APPENDIX

Underlying Cause of Death of 794 Persons Dying in Anchorage in 1985

CARDIOVASCULAR DISEASE				**219** (27.6%)
Heart Disease*			158	
coronary artery		135		
acute myocardial infarction	45			
hypertensive		7		
aortic valve stenosis		6		
congestive cardiomyopathy		4		
rheumatic		3		
type undetermined		3		

*excluding infections of the heart and deaths from heart disease before age 1

Vascular disease (other than coronary)			61	
cerebral thrombosis		27		
cerebral hemorrhage**		16		
aneurysm, Circle of Willis	8			
intraparenchymal	6			
subarachnoid, cause undetermined	2			
aortic aneurysm		7		
abdominal, ruptured	2			
abdominal, resected	2			
thoracic, dissecting	2			
thoracic, ruptured	1			
pulmonary embolism		5		
mesenteric artery thrombosis		3		
generalized atherosclerosis		2		
transposition of great vessels		1		

**excluding neonatal cerebral hemorrhage

CANCER		**198** (24.9%)
Lung	59	
Breast	22	
Colo-rectal	20	
Metastatic, origin undetermined	10	
Lymphoma	10	
Pancreas	8	
Brain	7	
Bladder	7	
Prostate	7	
Ovary	6	
Cervix	4	
Tongue	4	
Esophagus	4	
Stomach	3	
Kidney	3	
Myeloma	3	
Acute lymphatic leukemia	3	
Acute myelogenous leukemia	2	
Malignant melanoma	2	
Bile ducts	2	
Gallbladder	1	
Liver, primary	1	
Pharynx	1	
Larynx	1	

Embryonal cell, mediastinum			1	
Carcinoid			1	
Polycythemia rubra vera			1	
Pericytoma			1	
Penis			1	
Vagina			1	
Osteogenic sarcoma			1	
Sarcoma of bone, Ewing's			1	
VIOLENCE AND ADVERSE EFFECTS				**198** (24.9%)
Vehicular accidents			62	
automobile		45		
auto-pedestrian		8		
auto-bicycle		2		
auto-motorcycle		1		
3-wheeler		1		
bicycle		1		
train-pedestrian		1		
aircraft		3		
Alcoholism			36	
alcoholic liver disease		28		
acute alcoholism		5		
hypothermia		1		
acute pancreatitis		1		
lung abscess due to aspiration		1		
Suicide			35	
gunshot		17		
drug		7		
hanging		6		
carbon monoxide		3		
cut wrist		2		
Homicide			20	
gunshot		17		
assault	13			
accidental	3			
legal intervention	1			
bludgeoning		2		
method undetermined		1		
Miscellaneous			45	
fire		11		
fall, not on job		9		
on-job		8		
fall	4			
crush	3			
windblown object	1			
drowning		7		
drug overdose, accidental cocaine, heroin, or multiple drugs		5		
river accident, not drowning		2		
avalanche		1		
carbon monoxide, unknown whether accidental or suicide		1		
ingestion of insecticide, purpose undetermined		1		
DEATH BEFORE AGE 1				**51** (6.4%)
Congenital anomalies			19	
Immaturity			15	
Sudden infant death syndrome			13	

Cerebral hemorrhage			1	
Neonatal sepsis			1	
Psychomotor retardation			1	
Endocardial fibroelastosis			1	
INFECTION* **				**45** (5.7%)
Pneumonia			18	
Pyelonephritis			4	
Viral encephalitis			3	
Viral myocarditis			3	
Viral hepatitis			3	
hepatitis B		2		
non-A, non-B		1		
Acquired immunodeficiency syndrome			3	
Diverticulitis			3	
Meningitis, bacterial			2	
Epiglottitis, acute			1	
Endocarditis, bacterial			1	
Echinococcal abscess of liver			1	
Abdominal fistula, cause unknown			1	
Thrombophlebitis, septic			1	
Septicemia, source unknown			1	

***excluding neonatal sepsis

LUNG DISEASE				**38** (4.8%)
Chronic obstructive pulmonary diseases			33	
Asthma			2	
Restrictive lung disease due to tuberculosis			2	
Sarcoidosis			1	
NEUROLOGIC DISEASE				**17** (2.1%)
Alzheimer's disease			5	
Guillain-Barre' syndrome			3	
Senile dementia			3	
Chronic encephalopathy			3	
cause undetermined		2		
drug ingestion		1		
Parkinsonism			2	
Hydrocephalus			1	
MISCELLANEOUS				**28** (3.5%)
Diabetes mellitus+			5	
end stage renal disease		3		
insulin overdose, accidental		2		
Gastric ulcer			3	
bleeding		1		
perforated		1		
with acute respiratory distress syndrome		1		
Acute pancreatitis, nonalcoholic		3		
Aspiration of food		2		
Benign prostatic hypertrophy		2		
with obstructive uropathy	1			
with postoperative aspiration	1			
Systemic lupus erythematosus		2		
Hyperosmolar non-ketotic coma		1		
Hypothalamic-pituitary dysfunction		1		
Parathyroid adenoma with hypercalcemia		1		
Tear of trachea, cause undetermined		1		
Esophagogastritis, chronic, with inanition		1		

Gastrointestinal bleeding, cause undetermined	1	
Volvulus of colon	1	
Alpha-1-antitrypsin deficiency	1	
Aplastic anemia	1	
Decubitus ulcers	1	
Unexplained natural cause	1	
Total		794 (99.9%)++

+Diabetes mellitus was mentioned 29 times (3.7%) among 794 deaths.

++rounding artifact

ACKNOWLEDGMENTS

Daniel S. Janik, M.D., M.P.H., collected population data and assisted in calculations. Rose Courtright typed the manuscript.

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History of Medicine in Alaska

GEORGE HALE, M.D.

At seventy, George Hale is tall, trim and youthful looking. Many outdoor activities from hunting to running to skiing have helped him belie his years. It is only when he begins to tell of his various interests and pursuits that it becomes clear that these past years were lived in a full and productive life.

He was born in St. Louis, Missouri, the son of an American Baptist minister, on October 12, 1916. As a small child, George was seriously ill with pneumonia. He believes that the loving care of the physician who treated him influenced his decision to become a doctor.

While attending William Jewell College in Liberty, Missouri, from which he graduated *cum laude* in chemistry, George Hale considered becoming a medical missionary. He went to Harvard Medical School, earning an M.D. in 1943. He chose a rotating internship at Pennsylvania Hospital, Philadelphia, following it with a medical residency during which he also was an Instructor in Medicine at Jefferson Medical College.

During World War II, he served on the battle cruiser, U.S.S. Alaska, which was in the first all-Navy carrier raid on Tokyo. Later they sunk the Japanese super-battleship, Yamato, and aided the landings in Iwo Jima, Okinawa, Korea, and China. George Hale remained in the U. S. Naval Medical Reserve until 1955.

After the war, he resumed his training as a resident in Orthopedic Surgery and Instructor in Orthopedics at Albany Medical College. Following, he went to Washington, D.C. to spend two years as an assistant resident in surgery at the Washington Hospital Center.

Seeking a place to practice surgery in the private sector, he accepted a position with the Doctors Clinic in Anchorage. George remembered the stories he had been told by a residency roommate from Alaska about the wonderful hunting and fishing to be found. Never having been able to do enough of either, it was easy to make the decision. On July 2, 1949, after 19-½ hours of flying, the DC-4 landed at Anchorage.

George Hale was the thirteenth physician to begin practice in Anchorage and the only surgeon. He was called upon for all kinds of surgery from orthopedics to craniotomies. Starting as assistant chief surgeon for the Alaska Railroad in 1949, he became chief in 1951, serving in this capacity until he entered solo private practice of surgery in 1955.

Even with a busy practice and growing family, Dr. Hale found time to be involved in the growing community. He is still a member of the Board of Directors of the Alaska Division of the American Cancer Society, and he served on the Board of the American Cancer Society for 11 years. He was active in the establishment of the Alaska Mutual Savings Bank. In 1963, he was chief surgeon for the National Championship Alpine Race held at Alyeska.

George Hale was a member of the founding group of the Alaska State Medical Association and was its first president. He also helped found the Anchorage Medical Society and was president in 1957. He is a Fellow of the American College of Surgeons. His outstanding experiences are inevitably associated with the great outdoors: the pursuit of hunting, fishing, skiing, flying, and photography. He summarizes his avalanche encounter as follows: "Skiing from an airplane in the Alaska Range of mountains with my 12-year old son, John, . . . caught in an avalanche which swept us over a 50 foot cliff. John was buried. I broke four ribs and my back in the fall and was also buried except for my head. I dug out . . . found and dug my son out, and we skied three miles to safety." The complete account of this adventure can be found in *Alaska Magazine,* January 1980.

While strictly writing medical history, recounting the contributions of family may not be entirely appropriate; nonetheless, the impact of outstanding service to the state is an important part of the Hales' lives.

George and Mary Helen Parker were married in June 1942. Upon arrival in Anchorage, Mary taught choral music at the Anchorage High School. In the Spring of 1951, Mary Hale began directing the Community Chorus which she did for nine years. The Chorus appeared on the Ed Sullivan T.V. Show at the time of statehood, singing the Alaska Flag Song, and on Bing Crosby's Christmas Show.

Mary studied at San Diego State with Robert Shaw, whom she persuaded to come to Alaska, thus starting the Alaska Music Festival in 1956. The Festival began under the auspices of the Anchorage Community College, Anchorage Symphony, and the Community Chorus. Mary was its director for six years.

In 1966, Governor Egan appointed Mary to the first Alaska State Council of Arts, and in 1967, Governor Hickel appointed her chairman. For six years Mary Hale tackled this challenge—leaving to become an administrator at the Community College. Here, she began the Arts Affiliates Program, the popular "Fridays

10 til 2" program for women, and the Arts Fair attended each September by 35,000. Along the way, she induced the National Endowment for the Arts to establish the Alaska Humanities Forum.

Recently, she was appointed to the Executive Board of the Coalition of Community Colleges in Alaska which she serves as vice-president. In addition, she is involved with the Endowment Council at the Senior Center, American Association of University Women, Soroptimists International, and the Margaret Walkup Circle at the First Presbyterian Church.

The Hales had three children. John, who created and directed the first T.V. report on the Alaska Legislature and then served as aide to Senator Russ Meekins in Juneau, was killed in an airplane accident in July 1982 along with his wife, Deena, and their unborn child. Jim, a mountaineering guide and now pastor of the Talkeetna Christian Center, and Nancy Hale Lee both live in Talkeetna. There are five grandchildren.

On April 4, 1986 at midday, George Hale left his office after his last day of regular practice. There would be follow-up postoperative patients to care for, but this was retirement. He had practiced surgery in Anchorage for 37 years and there were 21 drawers of charts concerning thousands of people he had cared for during these years. The decision to retire didn't take long and wasn't difficult. With his many interests, retirement just presents new paths to follow. As he says, "Every day now is Saturday, and you can't beat that."

Interviewing was done by Marge Muzzall.

Gwynneth Gminder Wilson
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News from the American Society for Circumpolar Health

A Seminar Report: Medical Research in Chukotka

by Theodore A. Mala, M.D., M.P.H.[1]

On October 7, 1986, I attended a special seminar in Novosibirsk, U.S.S.R, on "Medical Research in Chukotka." Here follows a summary of the results of that seminar.

Chukotka covers an area of about 730 sq. kilometers in the far eastern part of Siberia. It is part of the Magadan administrative region and has a ratio of about one man for every three women in the Native settlements. The largest Native Group is the *Chukchii* which number about 14,000; the *Eskimos* with about 1500, and the *Lamouts*—but there were no statistics on them at hand. There are primarily *Eskimos* in the areas of Providenia and Uelen.

There are about 140,000 residents on the Chukchii Peninsula. The Institute of Internal Medicine in Novosibirsk studied primarily Natives and newcomers in 1979 and conducted about ten scientific expeditions to the area for the purpose of:

a) investigating the prevalence of cardiovascular diseases in Chukotka;
b) investigating the metabolism of lipids and carbohydrates;
c) investigating patterns of nutrition and social behavior including that of smoking.

Some of their findings are reviewed here.

In the area of *Risk Factors of Arterial Hypertension*, Siberian researchers found that 15% of Eskimo men between the ages of 30–59 had arterial hypertension, defined by standard WHO criteria as a systolic of 160 or greater and a diastolic of 95 or greater. In newcomers (non-Native) who had been in the same area for one year, 24% of the men they studied, aged 30–59, had arterial hypertension.

Another study examined *smoking habits* in men. A twelve-month study of those who smoked not less than one cigarette a day found that 67% of the Native men smoked compared to 63% of the male newcomers. Natives smoked an average of nine filtered and nonfiltered cigarettes per day compared to newcomers who averaged 18 cigarettes per day.

My Russian translator, Eugene Starkov, from the Praesidium of the Siberian Branch, Academy of Medical Sciences, told me a Russian story about statistics and how one must be careful with them. It goes like this: "If you have bacon and eggs for breakfast, and I have only a piece of dry toast, statistically, we both had a good breakfast!" The point of the story is that one must take the time to examine the surrounding facts before drawing too many conclusions from statistics alone.

A study of *obesity* was conducted. From a sample size of 450 Natives and 460 newcomers, using a standard body mass index of 29, 7% of the Natives were overweight compared to 28% of newcomers. Their occupations? Natives worked on collective farms and reindeer farms, while newcomers were employed in house building and as heavy equipment operators. The study sample was determined by the random selection of names from a list, and the study was done in spring (May).

Electrocardiographic studies were performed in the field using 12 channels and the Minnesota code—changes were detected in otherwise asymptomatic patients as a result of earlier scarring due to myocardial infarctions.

Eskimos were found to have a greater incidence of angina pectoris than Chukchii. Newcomers have about the same incidence as Chukchii. Left ventricular hypertrophy was found in 20% of Native men studied, some of whom showed no signs of hypertension. This ten-year study of about 80 Natives has not yet been published.

In summary, 9% less hypertension and obesity were found in Siberian Natives living in Chukotka than in newcomers to the area; the incidence of obesity was 21% less in Natives than in newcomers. Natives smoked two times less than newcomers. The incidence of angina was slightly less in newcomers than in Natives, and the incidence of cardiac hypertrophy—identified through electrocardiograms (EKG) in otherwise asymptomatic individuals—was 8% higher in Natives.

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[1] Dr. Mala is Associate Professor of Health Sciences, Researching Circumpolar Health Concerns, University of Alaska, Anchorage, 3211 Providence Drive, ADM 289, Anchorage, Alaska 99508.

Later expeditions followed up patients considered to be in high risk groups. Twenty of these were Chukchii identified with risk factors and signs of cardiomyopathy. They were followed the next year and found to be in good health, without any complaints and without evidence of arterial hypertension.

Methods of investigation included using A & B Mode Electrocardiography (using a Japanese monitor, Alooka SSD-260). Of 100 patients studied, some high risk patients were identified using both EKG and cardiography (ECHO); others were identified using one of the two diagnostic methods only. Investigators concluded that both EKG and ECHO must be used to identify all patients at risk. One method alone was found to be insufficient (Table 1).

Table 1. Methodology used to identify patients with cardiovascular high risk. Note 10 patients identified using one method only.

Number of Patients	EKG Only	ECHO Only	Both EKG & ECHO
Positive	Positive	Positive	Positive
100	10	10	80

Work was done on lipids utilizing the "Technitron" (USA) machine. Natives were found to have lower lipid and triglyceride levels than newcomers. Natives studied were primarily on "traditional diets" and newcomers were on "European" diets (Table 2).

Table 2. Lipid, Triglyceride and HDL levels in Chukota. Natives on Traditional Diets; Newcomers on European Diets.

	Natives (mg%/L)	Newcomers (mg%/L)
Lipids	196-198	210-212
(Based on 14-hr. fast)		
Triglycerides	76	116
HDL	60	51

Nutritional differences were also found between *sea coast Native residents* and *middle inland Native residents*. Sea coast Native residents were found to have less hypertension, decreased lipid levels, decreased body mass, lower coronary heart disease levels, and tended to live on a traditional diet. On the other hand, middle inland Native residents had health statistics that closely resembled those of the newcomers and were found to consume primarily a European-type diet.

To follow up these findings, a study was conducted in April and May of 1983 and 1984 of 270 Native men, 166 from sea coast areas (including 78 Eskimos) and 104 from tundra areas (Table 3).

Table 3. Diet intake in Siberian Natives both in sea coast areas (on traditional diets) compared to tundra area residents (on European diets).

Diet Content	Sea Coast Natives	Tundra Natives
Protein intake	32%	not measured
Kilocalories/day	3156	3000
Fat intake	36%	32%
Saturated fatty acids	11.5%	16%
Polyunsaturated fats	6%	2%
CHO intake*	26%	38%
*Refined sugar intake (%)	10% (of 26)	21% (of 38)
Ratio of saturated fats to poly-unsaturated fats	0.52	0.28

Another study examined 520 newcomers to Chukotka. Males were in the same age group, having lived in the Far North for about 11 years each: 25% were found to have arterial hypertension. All were either in the construction industry or working at the sea port (Table 4).

Table 4. Frequency of arterial hypertension by years of residence in the north. Sample = 520 males. Newcomers to Chukotka.

Years in North	Percent with Arterial Hypertension
1-3	7%
4-10	10%
11 and above	16%

Attempts to explain these findings include theories about the climate and the salt content of their diets. Using Fallers method of salt detection (based upon when an individual begins to taste salt in his mouth), an average of 0.16% was found in Chukotka newcomers compared to 0.13% in residents of Novosibirsk. Does increased salt usage necessarily lead to increased blood pressure? Salt intake was found to be increased in newcomers to Chukotka compared to salt intake in Novosibirsk.

Researchers attempted to further examine differences between Natives and newcomers in respect to diet and lipid levels. Since Natives were found to have lower triglycerides, the question was, "Is there a genetic protective factor?" A group of 772 Natives was examined, half Chukchii and half Eskimo, ranging in age from 6 to 72 years. An *age variability* produced certain trends. Different from other populations examined, it was found that in Natives, triglyceride levels *decreased* with age, while HDL levels *increased*. The greatest changes occurred between the ages of 15 to 45 years. HDL *increased* with age in males and *decreased* with age in females.

Among families of patients in high risk groups, increased lipid levels and hypertension were present, quite possibly linked to genetic factors. Investigators hypothesized that the gene role of the x-chromosome is less than in that found in larger populations.

Further studies in boarding schools were conducted. Of 300 students, 200 were either Eskimo or Chukchii between grades 6-12. All students were fed a European diet. Risk factors in both groups were the same, but incoming Native children at the time of first admission to school had lower lipid levels than their peers. First signs of obesity showed up earlier in Native children than in others.

Finally, testing was done for diabetes mellitus in Natives and newcomers. Subjects studied using glucose tolerance tests were between the ages of 30 and 59. There was a difference in enzyme metabolism between the two groups. There were no changes in Natives, but newcomers had enzyme levels three times higher than the Native group. Basal insulin levels were decreased in Natives in fasting states as compared to newcomers (Table 5). Investigators wanted to study the role of insulin, cortisone, and human growth factors.

Table 5. Comparisons in fasting levels between Siberian Natives and Siberian newcomers.

	Natives	**Newcomers**
Cortisone		increased
Basal level insulin		increased
Growth hormone	same	same

Investigators believe that increased cortisol levels result in increased incidence of diabetes mellitus, and, in future studies want to examine the relationship of glucose, cortisol and glucose tolerance disturbances through long-term prospective studies.

Dr. Diana Feizuchanova, Chief of Pediatrics at the Institute of Internal Medicine in Novosibirsk, has done an extensive study of 2200 children between the ages of 10-14 years examining the size of shoulders, chest, height, weight, and lipid, triglyceride, and HDL levels. Her study included questions about smoking, physical activity, family history and maturation. A special thiocyanate test was done (using WHO standards) to test for the presence of nicotine. The study (not yet published) compares Siberian children to those of Moscow (Table 6).

Table 6. Smoking habits in Siberian boys by age.

Age	**Percent Smoking**
> 11	8%
> 14	20-25%

Siberian findings reveal average cholesterol rates (even decreased in boys) and decreased amounts of lipids but not of HDL. Girls above 14 years of age were found to have values closer to that of women.

Triglyceride levels were similar to US students, but cholesterol levels were *increased* in Siberian girls by 10-15 mg./L. Cholesterol levels for boys were the same as US levels. HDL levels were higher in Novosibirsk than in Moscow. It is hoped that the same study can be done between Alaska and Siberia.

A study of Alaskan-Siberian nutrition habits would be of value in determining the role of nutrition to disease. The proposed Alaskan Siberian study would take place in Siberia between Novosibirsk and Chukotka using WHO methods and standards. Nutrition is thought to plan a key role in the prevention of many diseases in the north, especially in the intake of saturated fatty acids. The problem should be studied from the point of view of cardiovascular diseases, diabetes, obesity and anthropological considerations.

Also proposed are joint studies to examine the role of cold, stress and the fundamental aspects of adaptation to the North. Anadir is the main city of Chukotka with a population of 12,000 people. The rest of Chukotka is mostly rural or "bush" in nature. Studies would be conducted in both as well as compatible locations in Alaska.

CONCLUSIONS

There is much that can be learned from Siberia and Alaska. The two neighbors across the Bering share much in common in history, ethnography, and development. It has been too many years since regular communication has flowed between the two. Now we are presented with the opportunity to do something about it through the recently signed Alaskan-Siberian Medical Research Exchange Program at the University of Alaska.

ACKNOWLEDGMENT:

Special thanks is given to Dr. Yuri P. Nikitin, Director of the Institute of Internal Medicine in Novosibirsk, for arranging this seminar to acquaint western colleagues with some of their research work.

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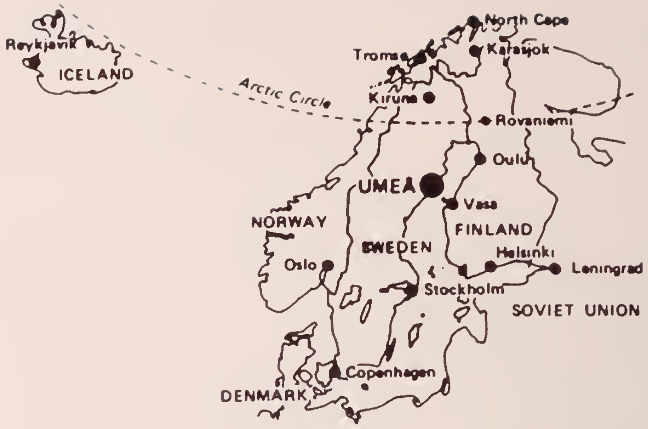

Preliminary list of principal speakers.

Honorary lecturer:	Professor Carleton Gajdusek, Nobel prize winner 1976.
K. Rodahl, Oslo:	Occupational health in circumpolar regions.
P.J. Blind, Umea:	Lapp society yesterday and today — a comparison.
M. Freeman, Edmonton:	Environment society and health: An examination of quality of life issues in the contemporary North.
A. Forsdahl, Tromso:	From poverty to prosperity — hazard to the health of the people in the Arctic?
Yu. Borodin, Novosibirsk:	Investigations by Siberian scientists in the North.
B. Postl, Winnipeg:	Assumption of control and health in Canadian native populations.
D.J. Lugg, Kingston, Australia:	Antarctica — lessons for health care planning and delivery to circumpolar populations.
W.W. Myers, Seattle:	Who cares? Training health personnel for disadvantaged communities.
I. Waddington, WHO, Copenhagen:	Environmental health considerations in the Arctic.
R. Shephard, Toronto:	Cold adaptation and energy balance.
P-O. Granberg, Stockholm:	Cold injury in clinical practice.
A. Eriksson, Amsterdam:	Aspects of genetic epidemiology of the North Calotte.
K. Young, Winnipeg:	Chronic diseases among Canadian Indians.
T. Sjovold, Stockholm:	Skeletal evidence of colonization, contemporary habitation and health to the north of the arctic circle in the past.
G. Nikiforuk, Toronto:	Aspects on dental health in remote areas.
J.H. Weisburger, New York:	Comparison of nutrition as customery in the Western World, the Orient, and Northern populations (Eskimos) in relation to specific disease risks.
H. Forsius, Oulu:	Astigmatism and myopia in arctic regions.
M. Anniko, Umea:	Clinical and morphological findings in respiratory epithelia in some patients from Northern Sweden.
H.K. Akerblom, Helsinki:	Coronary heart disease and risk factors in circumpolar areas.
Yu. Nikitin, Novosibirsk:	Chronic noninfectious disease in native and new-coming populations in circumpolar areas.
J.P. Hart Hansen, Copenhagen:	Circumpolar cancer — etiology and prevention.

Main Topics of the Congress

1. Occupation and health in circumpolar area.
2. Environment in the Arctic and Subarctic.
3. Health care planning and delivery, vital statistics.
4. Health care education.
5. Health care engineering.
6. Industrial development and health.
7. Cold injury, hypothermia; adaption, physiology and pathology in cold climates.
8. Genetics and epidemiology.
9. Anthropology and demography.
10. Oral health.
11. Nutrition.
12. Diseases of the eye, ear, nose and throat.
13. Infectious diseases.
14. Cancer and other (chronic) diseases.
15. Mental health, alcoholism, drug and child abuse.
16. Emergency medical services and communication.
17. Biological and circadian rhythms.
18. Arctic medical history and traditional medicine
19. Maternal and child health.

Workshops, round table discussions, panels, symposia, seminars, study groups.

1. Arctic Research — what and how?
2. Community planning and housing in the Arctic (panel).
3. Hepatitis B.
4. Association of HLA — B 27 with reumatoid arthritis in Nordic populations.
5. Radioactive fallout and health effects.
6. The endocrinology of cold environment.
7. Study groups meeting on polar cancer.

For local information call:
John P. Middaugh, M.D.
(907) 561-4406

efore prescribing, see complete prescribing information in SK&F CO. terature or *PDR*. The following is a brief summary.

WARNING

This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

ontraindications: Concomitant use with other potassium-sparing agents uch as spironolactone or amiloride. Further use in anuria, progressive enal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum otassium. Hypersensitivity to either component or other sulfonamide-erived drugs.

Varnings: **Do not use potassium supplements, dietary or otherwise, nless hypokalemia develops or dietary intake of potassium is markedly npaired.** If supplementary potassium is needed, potassium tablets hould not be used. Hyperkalemia can occur, and has been associated ith cardiac irregularities. It is more likely in the severely ill, with urine olume less than one liter/day, the elderly and diabetics with suspected r confirmed renal insufficiency. Periodically, serum K$^+$ levels should be etermined. If hyperkalemia develops, substitute a thiazide alone, restrict $^+$ intake. **Associated widened QRS complex or arrhythmia requires rompt additional therapy**. Thiazides cross the placental barrier and ppear in cord blood. Use in pregnancy requires weighing anticipated enefits against possible hazards, including fetal or neonatal jaundice, hrombocytopenia, other adverse reactions seen in adults. Thiazides ppear and triamterene may appear in breast milk. If their use is essential, he patient should stop nursing. Adequate information on use in children s not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or ctivation of systemic lupus erythematosus has been reported with hiazide diuretics.

recautions: The bioavailability of the hydrochlorothiazide component of Dyazide' is about 50% of the bioavailability of the single entity heoretically, a patient transferred from the single entities of triamterene nd hydrochlorothiazide may show an increase in blood pressure or fluid etention. Similarly, it is also possible that the lesser hydrochlorothiazide ioavailability could lead to increased serum potassium levels. However, xtensive clinical experience with 'Dyazide' suggests that these conditions ave not been commonly observed in clinical practice. Angiotensin-onverting enzyme (ACE) inhibitors can elevate serum potassium; use vith caution with 'Dyazide'. Do periodic serum electrolyte determinations particularly important in patients vomiting excessively or receiving arenteral fluids, and during concurrent use with amphotericin B or orticosteroids or corticotropin [ACTH]). Periodic BUN and serum reatinine determinations should be made, especially in the elderly, iabetics or those with suspected or confirmed renal insufficiency. umulative effects of the drug may develop in patients with impaired renal unction. Thiazides should be used with caution in patients with impaired epatic function. They can precipitate coma in patients with severe liver isease. Observe regularly for possible blood dyscrasias, liver damage, ther idiosyncratic reactions. Blood dyscrasias have been reported in atients receiving triamterene, and leukopenia, thrombocytopenia, granulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes nellitus. The effects of oral anticoagulants may be decreased when sed concurrently with hydrochlorothiazide; dosage adjustments may be ecessary. Clinically insignificant reductions in arterial responsiveness o norepinephrine have been reported. Thiazides have also been shown to ncrease the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic lood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with Dyazide' The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function. Thiazides may add to or potentiate the action of other antihypertensive drugs. Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

Supplied: 'Dyazide' is supplied as a red and white capsule, **in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.**

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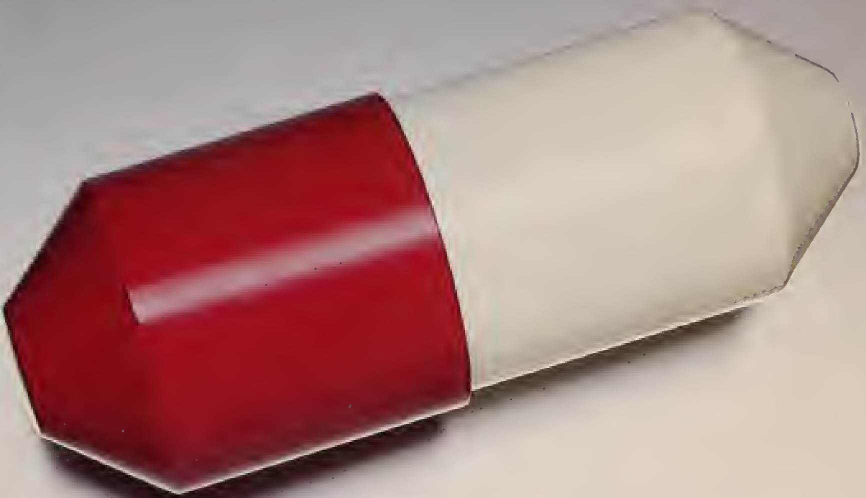

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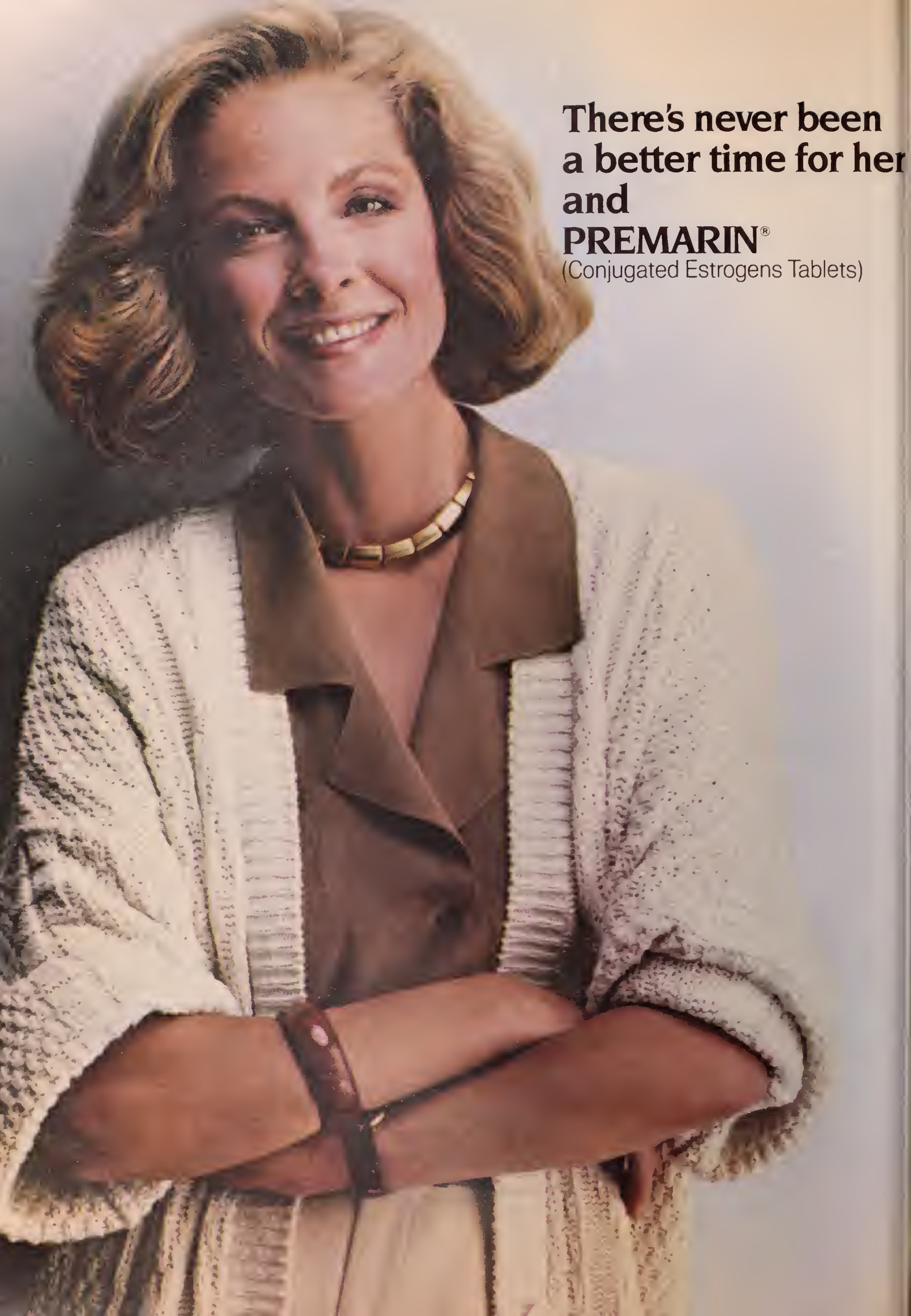
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Significantly reduced risk of endometrial hyperplasia

Endometrial hyperplasia was significantly reduced when progestin was added to PREMARIN therapy for more than ten days a month.[1-4] The risk of endometrial hyperplasia may also be reduced through cyclic administration of unopposed, low-dose PREMARIN.

Effect on lipids—an important feature

PREMARIN used alone does not adversely affect lipid levels. In fact, a clinical study has shown a significant increase in HDL cholesterol—from 49.7 mg/dL to 56.4 mg/dL—and decrease in LDL cholesterol—from 165.1 mg/dL to 138.1 mg/dL—after one year of therapy with PREMARIN, 0.625 mg.[5]

Low-dose control of menopausal symptoms*

PREMARIN effectively relieves vasomotor symptoms, such as hot flashes. When estrogen deficiency is limited to atrophic vaginitis, PREMARIN® (conjugated estrogens) Vaginal Cream restores the vaginal environment to its premenopausal state.

The most widely used, most extensively studied estrogen worldwide.

PREMARIN®
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Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

For moderate-to-severe vasomotor symptoms

PREMARIN®

(Conjugated Estrogens Tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

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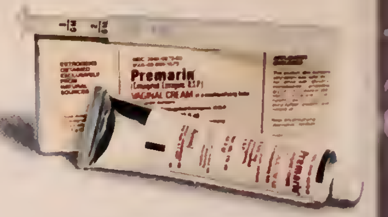
For atrophic vaginitis

PREMARIN®

(Conjugated Estrogens)

Vaginal Cream

0.625mg/g



BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base

1 ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA

Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13.9 times greater than in nonusers The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses

2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.

The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4.7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0.3 mg, 0.625 mg, 0 9 mg, 1 25 mg, and 2.5 mg strengths of conjugated estrogens Cream is available as 0 625 mg conjugated estrogens per gram

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass)**. Atrophic vaginitis Kraurosis vulvae Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (See Boxed Warning) 4 Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy)

WARNINGS: Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease, or abnormal mammograms A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrog per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis When doses of this size used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tendern abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estro containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observe patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be care observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastase

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic org and should include a Papanicolaou smear As a general rule, estrogen should not be prescribed for longer one year without another physical examination being performed Conditions influenced by fluid retention suc asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleed mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increa incidence of mental depression Patients with a history of depression should be carefully observed Preexis uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estro therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, medication should be discontinued while the cause is investigated Estrogens should be used with care in pati with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, young patients in whom bone growth is not complete If concomitant progestin therapy is used, potential may include adverse effects on carbohydrate and lipid metabolism

The following changes may be expected with larger doses of estrogen:

a Increased sulfobromophthalein retention

b Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3; increased epinephrine-induced platelet aggregability

c Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone measured by PBI, T4 by column, or T4 by radioimmunoassay. Free T3 resin uptake is decreased, reflectin elevated TBG, free T4 concentration is unaltered

d Impaired glucose tolerance

e Decreased pregnanediol excretion

f Reduced response to metyrapone test

g Reduced serum folate concentration

h Increased serum triglyceride and phospholipid concentration. As a general principle, the administratio any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in hu milk

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contracept breakthrough bleeding, spotting, change in menstrual flow; dysmenorrhea; premenstrual-like syndro amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, chan cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secr (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma w may persist when drug is discontinued, erythema multiforme, erythema nodosum; hemorrhagic eruption; lo scalp hair; hirsutism, steepening of corneal curvature; intolerance to contact lenses; headache, migra dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance; aggr tion of porphyria; edema; changes in libido

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only.* For treatment of moderate to severe *vasomotor* symptoms, atro vaginitis, or kraurosis vulvae associated with the menopause (0.3 to 1.25 mg or more daily) The lowest dose will control symptoms should be chosen and medication should be discontinued as promptly as poss Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or t medication should be made at three- to six-month intervals

2 *Given cyclically*: Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically A upward or downward according to response of the patient. For maintenance, adjust dosage to lowest leve will provide effective control. Osteoporosis —0.625 mg daily. Administration should be cyclic (eg, three w on and one week off).

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate meas taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only. For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinue promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recu abnormal vaginal bleeding

References:

1. Whitehead MI, Townsend PT, Pryse-Davies J, et al: Effects of estrogens and progestins on the biochemistr morphology of the postmenopausal endometrium *N Engl J Med* 1981;305:1599-1605 **2.** Paterson MEL, W Evans T, Sturdee DW, et al: Endometrial disease after treatment with oestrogens and progestogens in climacteric. *Br Med J* 1980;280:822-824 **3.** Magos AL, Brincat M, Studd JWW, et al: Amenorrhea endometrial atrophy with continuous oral estrogen and progestogen therapy in postmenopausal women. *O Gynecol* 1985; 67: 496-499. **4**. Whitehead MI, Lane G, Siddle N, et al: Avoidance of endometrial hyperstimul in estrogen-treated postmenopausal women. *Semin Reprod Endocrinol* 1983;1:1;41-52. **5**. Barnes RB, R Lobo RA. Comparison of lipid and androgen levels after conjugated estrogen or depo-medroxyprogeste acetate treatment in postmenopausal women *Obstet Gynecol* 1985;66:216-219

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"Living in the city
is lonely enough...
with herpes it's like
solitary confinement."

ZOVIRAX®
(acyclovir)
CAPSULES

Prevent genital herpes recurrences month after month with daily therapy.

(In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.)

Please see last page of this advertisement for brief summary of prescribing information.

ZOVIRAX® (acyclovir) CAPSULES

Help free your patients from recurrences.

Daily therapy

Coping with genital herpes is rarely easy. For some, the worst part is the pain and discomfort of frequent attacks—month after month, year after year. For others, the emotional burden presents a more difficult problem, leading to social isolation, anxiety, and diminished self-esteem.

Prevent or reduce recurrences

Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

Generally well tolerated

Daily therapy with ZOVIRAX CAPSULES is generally well tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/vomiting, vertigo, and arthralgia.

The physical and emotiona difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episodes as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRA CAPSULES.

Please see brief summary of prescribing information on next pag

Prevent recurrences month after month*

ZOVIRAX®

(acyclovir)

CAPSULES

Brief Summary

INDICATIONS AND USAGE: Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psychosocial difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

First Episodes (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

Recurrent Episodes:

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

CONTRAINDICATIONS: Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

WARNINGS: Zovirax Capsules are intended for oral ingestion only.

PRECAUTIONS: General: Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

Drug Interactions: Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the F_1 generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

Pregnancy: *Teratogenic Effects:* Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS — Short-Term Administration: The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

Long-Term Administration: The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

Chronic suppressive therapy for recurrent disease: One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

Intermittent Therapy: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

Patients With Acute or Chronic Renal Impairment: One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance ≤10 ml/min/1.73/m².

HOW SUPPLIED: Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200"- Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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*In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

Wellcome

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Arctic's Mysteries: Coming in from the Cold

by Linda Bosy, reprinted by permission of American Medical News

The arctic's many mysteries may start to unfold in 1987 as provisions of the Arctic Research and Policy Act are set on a path toward implementation.

This year will be a pivotal one in the organization of a broad-based, coordinated federal and state research program in which five years of work in the areas of health, environmental protection, weather, communication, transportation, and resource development will be outlined.

Already, much work toward development of a five year plan, as ordered in a 1984 federal law, has been accomplished following an exhaustive multi-agency assessment of national arctic research needs.

In mid 1986, the U.S. Arctic Research Commission, composed of presidential appointees and one of two steering groups called for in the legislation, issued a report titled "A Framework for Action."

The report stated that the basic premise of arctic research should be comprehensive, interdisciplinary, and coordinated for acquiring the scientific and engineering knowledge for national security and defense, resource development, and well-being of the population.

Among the report's highlights were a number of research program suggestions:

- To try to understand the Arctic Ocean and how it, with the atmosphere, operates as a coupled component within the arctic system.
- To try to understand the high-latitude upper atmosphere and its extension up into the magnetosphere.
- To consolidate arctic data and information through a transfer system.

Most of the high-priority research that the commission recommended, however, is based on the area's health-culture-socioeconomic system to "identify and resolve the major health, behavioral, and cultural problems that derive from distinctive characteristics of the arctic environment."

For example, the commission called for research on the health and adaptation of arctic inhabitants to cold and the effect on people of resource development and industrial growth and urbanization.

Even for personnel conducting much of the non-health-related research in the arctic, the issues of adaptation to unusual environment such as cold, darkness, and isolation, and the stressful situation of long hours and six-week shifts need to be studied.

To further define the health aspects of the arctic research program, a 12-member Alaska State/Federal Health Task Force was created, co-chaired by John Middaugh, MD, Alaska State Health Department epidemiologist, and George F. Hardy Jr., MD, of the Centers for Disease Control.

That task force identified three national areas of research priority: intentional and unintentional injuries; diet and atherosclerosis; and cancer.

"Our criteria for identifying research areas are that they must be of national importance, have been neglected in the past, show evidence of ongoing federal and state cooperation, can be accomplished if enhanced resources were available, and be in areas where their scientific merit pass peer review," Dr. Middaugh explained.

Future priority research areas will be for infectious diseases and improved international health research collaborative efforts, Dr. Middaugh added.

"We are really excited to have all these opportunities," Dr. Middaugh told AMN, "since one example after another has shown that arctic research can have national significance, such as our research on hepatitis B and Hemophilus influenzae disease."

In addition, research on portable lasers was conducted in remote arctic areas by researchers from Johns Hopkins U., Dr. Middaugh said, showing that glaucoma patients in remote areas can undergo eye surgery.

Dr. Middaugh praised the American Medical Association and the American Public Health Association for their lobbying efforts to include health as a priority in the arctic research bill. "Health research here has universal potential [application]," he added.

Currently, at least 11 federal agencies, under separate budgets, have ongoing arctic research programs into national security, resource development with minimal environmental and social impacts, and "natural laboratory" experiments that look at Native culture and wildlife habitats.

The idea behind the law is to consolidate all of those individual efforts into a coordinated one, said Dr. Hardy, another member of the state/federal task force.

"Arctic" under the public law means all United States and foreign territory north and west of the boundary formed by the Porcupine, Yukon, and Kuskokwim rivers; all contiguous seas; including the Arctic Ocean and the Beaufort, Bering, and Chukchi seas; and the Aleutian Chain.

Two-and-a-half years after the law's enactment, with committees intact, periodic public workshops, and private reviews, it appears likely that the Arctic Research Commission's detailed five-year plan could be presented to Congress for funding approval sometime this year.

If Congress approves the plan, it would be included in the fiscal 1989 budget, said Lyle Perrigo, the commission's staff officer.

Dr. Hardy, however, who is in the process of revising another draft of the task force's plan, did not offer that same timetable, although he did say that the "health portion of the plan is in fairly good shape."

A Vital Call for Physician Cooperation

In barely four years since it was first identified and named, AIDS—Acquired Immunodeficiency Syndrome—has become one of the most serious epidemics this country has ever seen. Without exaggeration, it can be said that AIDS threatens to become the major international lethal pandemic of the twentieth century.

More than 20,000 cases have been found in this country; thousands more around the world. More than half of those who have contracted it have died. Estimates by medical and scientific experts who are studying AIDS are that probably two million people in the United States have already been infected with the virus, HTLV-III, now called HIV. The Centers for Disease Control predicts 274,000 cases of AIDS by the year 1991, of which about half will be fatal. And the CDC says its predictions may actually underestimate the situation.

Doctors across the country are being asked to cooperate in bringing accurate information about the disease to their patients and to the public at large. They are also being asked to cooperate in another pressing effort: notifying their patients who have received blood, plasma or platelets from sources that could have been tainted with the virus long before the blood banks had an available test for HIV among their donors.

The American Red Cross, the American Association of Blood Banks and the Council of Community Blood Centers are working together on a look back program, checking previous donation records of all would be donors who now test positive for HIV. If it is found that such a person has donated before (an estimated 85 percent of the 12 million annual blood donations are from previous donors), all patients who have received the possibly contaminated blood or blood products will be identified, and hospitals or other institutions where the transfusions were given will be notified. They, in turn, will be asked to notify you, if one of your patients was involved.

Your first responsibility is to notify the patient that, although the odds are very slim, he or she might have been exposed to the virus. You should then refer the patient to a local testing center.

The look back program has been designed in a way that will maximize the chance of finding those few persons who may have been infected (and can possibly spread the virus), while at the same time, only inducing anxiety in a small number of persons. While we are distressed at the emotional impact this outreach can have, we feel this is a vital way we, as individuals, can actively curb the spread of AIDS in our communities.

President's Page

Lobbying—a dirty word? A necessary function in a democracy? Often it seems to be somewhere in between. The Alaska State Medical Association has been deeply involved in lobbying during the last two legislative sessions because of the major thrust toward Tort Reform. It dawned on me of late that some of our membership in certain specialities may feel that other issues have been slighted. I think that's an understandable feeling. Tort Reform is a major nationwide issue, deservedly receiving a lot of attention because it appears there is a "window of opportunity" to obtain some significant changes in our society's means of redressing grievances. I believe that it is worth the emphasis it has received and will continue to be worth that until we achieve substantial success or until we decide in our best judgment that we have done all that we can and should move on to other matters.

However, in the meantime other issues have not gone away. For instance, it is a high priority of our Council to obtain passage of legislation assuring adequate funding for the State Medical Board. By the time you read this, I sincerely hope that such passage will have occurred, but if it has not, you may be assured we are still working on it. Other issues such as the effort of optometrists to obtain diagnostic and therapeutic drug privileges that have traditionally and appropriately belonged to ophthalmologists, and perhaps other specialty interests that I do not yet know about, continue to receive our attention. In the heat of the Tort Reform battle, I fear some issues may have received less attention than they deserve. To help correct this, we have recently instituted a Legislative Committee which serves in an advisory capacity to the President, the Executive Director, the Legislative Coordinator, and our lobbyist in an attempt to achieve a broader and more balanced view of legislative issues and to be sure that more points of view are represented.

If you, your specialty society, or your locality feel that you have important legislative issues that need support, you must "stick your oar in the water." Every other week during the legislative session we have a conference call meeting of the Legislative Committee. Those with an intense interest in a medical issue are invited and urged to notify our Executive Director and ask to be included in that conference call. Of course, it remains Association policy that the Council makes legislative policy. However, it is very common that issues have already been considered by the Council in past years, and the concerns of members fit right in with previous ASMA policy. In that circumstance, all that is needed is guidance to our Legislative Coordinator and lobbyist so that proper emphasis can be given to lobbying for those issues.

Each of us has issues that are nearer and dearer to our hearts than others, and the issue of Tort Reform has been among the nearest and dearest for those of us closely involved in the legislative process the past two years. However, we also recognize an obligation to fairly represent our membership, and we genuinely want to hear from you if you have important issues.

Donald E. Thieman, M.D.
President
Alaska State Medical Association

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REGISTRATION/ INFORMATION

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Anchorage, Alaska 99508
Phone (907) 562-2662

Registration Fee: $250.00 for Non-Member physicians. This fee entitles physicians to attend all CME sessions and activities. No fee is required for ASMA members.

LOCATION & DATE OF MEETING

Centennial Hall Convention Center
Juneau, Alaska
(907) 586-9442
June 3 - 7, 1987

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A full slate of spouse and children's activities are being planned by the ASMA Auxiliary. Babysitting will be available.

TRANSPORTATION

Alaska Airlines provides daily jet service from Seattle, Anchorage, and other Alaskan cities. Phone 800 426-7464 for reservations.

Ferry service is available from Seattle and S.E. Alaska. Phone (907) 272-4482. Shuttle, taxi service, and rental cars are available at the Juneau airport.

Anchorage Air Charter

ASMA member physicians and their families are invited to board the chartered 737 "Fun Flight" to Juneau for the incredible price of $184 round trip.

For non-member physicians and families a special price of $229 is available.

DEPOSIT/CANCELLATION

Physicians who take advantage of ASMA's low cost "Fun Flight" must send full payment by April 1, 1987. (Non-refundable)

Non-member physicians must send $50 pre-registration fee by April 15. Full refunds will be made upon request prior to May 1, 1987. No refund thereafter.

SCHEDULE OF EVENTS

Wednesday – June 3, 1987

8:00 - 12:00 noon
ASMA Council Meeting & Business Session

1:00 - 4:30 p.m.
Scientific Session

5:00 - 6:00 p.m.
ASMA's Reception for Exhibitors

6:45 - 9:30 p.m.
Dinner with Entertainment
Russian Dancers

Thursday – June 4, 1987

8:00 - 9:30 a.m.
ASMA Business Meeting

10:00 - 12:00 Noon
Scientific Session

12:00 Noon
Lunch at Convention Center
Guest Speaker - "History of Russian Orthodoxy in Alaska"

1:30 - 4:15 p.m.
Scientific Session

5:30 - 8:00 p.m.
Dinner at Thane Ore House
"Gold Nugget Review"

Friday – June 5, 1987

7:30 - 9:00 a.m.
Specialty Breakfast Meetings

10:00 - 12:00 Noon
Scientific Session

12:00 Noon
ALPAC Luncheon - Centennial Hall

1:00 - 2:00 p.m.
ASMA Business Session

2:30 - 8:30 p.m.
Glacier Trip Dinner Cruise

Saturday – June 6, 1987

8:00 - 9:30 a.m.
"Fun Run" with Jim Ryan

10:00 - 12:00 Noon
Scientific Session

12:00 Noon
Lunch sponsored by Syntex

1:30 - 4:45 p.m.
Scientific Session

6:00 p.m.
President's Reception & Dinner
Dance with Band
Installation of Officers, Awards

Sunday – June 7, 1987

9:00 - 11:00 a.m.
ASMA Council Meeting

REGISTRATION FORM

NAME: DATE:

ADDRESS:

ZIP:

NUMBER OF ADULTS IN PARTY:

SPOUSE'S NAME:

NAMES & AGES OF CHILDREN: AGE:

ANCHORAGE AIR CHARTER – NUMBER IN PARTY:

Send check or money order with Registration Form to: **Alaska State Medical Association**
2401 E. 42nd Avenue, Suite 104 Anchorage, AK 99508

Executive Privilege

During the First Continental Congress, Virginia statesman, Patrick Henry, strongly felt that the states should go it alone. But in hindsight, we now know that decision would have spelled disaster.

The situations and circumstances facing our physicians in Alaska are not unlike the problem that our founding fathers faced in creating a "united states". They, too, were faced with the decision of becoming a "united states" or continuing independently to face the challenges from the outside that would affect their commerce and their ability to control their own destiny. Should physicians "go it alone" or should they attempt to become "united" in their efforts against outside forces? That is the question that is before us now.

As the director of your association, I have attempted to become informed with the issues over the past six months. I agree, not much time to become an expert, but I believe time enough to understand the forces that will affect your practices. Therefore, I believe it is my responsibility to speak out on the issue of an IPA.

In my judgment, I believe that the physicians of Alaska are faced with the same dilemma as Patrick Henry. I am also convinced that the greatest threat comes from the same body that Patrick Henry feared, government itself. The U. S. Government Office of Management and Budget has proposed expanding the DRG system to include physician payment, the CHAMPUS program has now entered the bidding arena, the Veteran's Administration has made overtures to ASMA to control costs, and the continued instability of Medicare and Medicaid all demonstrate convincingly our government's increasing attempts to control health care.

Many of you have expressed concern and dismay that your independence will be threatened. Yes, the formation of any organized association may do that. However, will you have the power in the future to dictate your level of income, the standards and quality of care? I suspect not. We cannot be lulled into believing that the forces that affect your colleagues in the lower forty-eight will not affect you. But the formation of an organized independent practice association will give you the unified power to keep the decision-making process where it belongs . . . with Alaskan physicians. In the midst of controversy and termoil, keep in mind that the process and opportunity for dissent is an essential ingredient in obtaining the facts necessary to make decisions.

Just like Patrick Henry and the founding fathers, your decision will not only affect the welfare to your patients, but the direction of medicine in Alaska for years to come.

Raymond Schalow
Executive Director
Alaska State Medical Association

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Auxiliary News:

Alaska State Medical Association Auxiliary

To update you, near and far, on your state auxiliary's activities I'd like you to know four items:

1. ASMA Auxiliary contributed over $7,000 to keep the American Lung Association's Asthma "Champ Camp" financially afloat this coming summer. Members-at-large throughout Alaska have been contacted about contributing. This is still pending. The Lung Association awarded us a citation recognizing the largest monetary contribution to fight lung disease in the state. I was awarded the double barred cross for organizing the raffle which was so successful.
2. We have sent in over $2,000 to AMA-ERF Scholarship Fund, and we are not done with this project.
3. I am working concomitantly with Ray Schalow and his staff to offer you a stimulating and memorable convention.
4. ASMA is joining efforts with the Anchorage Medical Society Auxiliary to assist in the Children's Healthfest. This should result in a positive experience for the little participants and their families, and boost the image of the physician in the community.

Until the next issue, I am your ASMA-Auxiliary president,

Patrice Gerster
President, ASMA Auxiliary

Anchorage Medical Society Auxiliary

The Anchorage Medical Society Auxiliary has begun to gear up for the major project of the year—*Children's Healthfest '87*. This will be a health fair for children, held in the Northway Mall on April 11.

It will fulfill three goals:

1. to promote good health practices, good health awareness and healthful life-styles in children,
2. to benefit the children and families from all segments of the Anchorage community, and
3. to improve the image of the entire medical community in Anchorage.

We hope to have nearly every medical specialty represented in booths, special events, or the "mock-up hospital" including an OR, ER, X-Ray, cast room, doctor's office, etc.

The auxiliary has been active in a variety of other projects this winter. The "Totally Tacky and Tasteless Trot" was held in October and got rave reviews. It netted $1,650; $200 went to AMA-ERF, and the rest to the Children's Healthfest. In November the auxiliary participated in the United Way drive by delivering United Way pledge packets to all the health professionals in Anchorage and then picking up the pledges. The Christmas Social was held in December, at which time gifts were collected for the occupants of Providence House. The auxiliary also organized the Medical Society Christmas Brunch where the AMS officers were installed. The bylaws revision, an ongoing project for the last several years, was finalized, sent to members, and approved. A Marianne Wieland print was advertised nationally in "Alaska Magazine", and we received orders totalling over $700—profits which benefit the scholarship fund. The January meeting was combined with the AMS meeting and focused on IPA's. We are currently planning a family ski evening and potluck at Kincaid Park to be held in late February.

The AMS auxiliary is an active, interesting group—think about getting involved with us.

Alice Samuelson
President, AMS Auxiliary

Editorial

With this issue we welcome the members of the American Society for Circumpolar Health who have selected *Alaska Medicine* as their official scientific journal. All Society members will now receive *Alaska Medicine*, beginning with this issue. The American Society for Circumpolar Health was founded in 1967 by then Commissioner of Health, Earl Albrecht, M.D. The purposes of the Society are to:

- serve as a vehicle for maintaining communication among people interested in all aspects of human health in the circumpolar regions of the world;
- promote public awareness of the current situation of circumpolar health;
- encourage and support human health-related scientific research and education in the circumpolar world; and
- serve as the vehicle for assuring U.S. attendance at international circumpolar health symposia.

Under Dr. Albrecht's leadership, the Society was instrumental in establishing the International Symposia on Circumpolar Health, held every three years since 1967. The 6th International Symposium was held in Anchorage in May 1984, drawing 770 registered participants from 22 countries. In June 1987, the 7th Congress will take place in Umea, Sweden, hosted by the Nordic Council for Arctic Medical Research. The program will be of great interest to all Alaskan physicians. There is still plenty of time to register and participate in the Congress and post-Congress special travel and scientific programs. We are anticipating an added benefit—we hope to publish many of the scientific papers that will be presented at the Congress. I urge you to join the American Society for Circumpolar Health—participate in international sharing of medical knowledge and explore knowledge from polar regions. A membership application and information on registration for the 7th International Congress are to be found elsewhere in this issue of *Alaska Medicine*.

We are continuing our efforts to make *Alaska Medicine* an essential part of your reading list. Let us hear from you—submit an article, case-report, editorial, or letter to the editor. My thanks to all who responded to my request for reviewers of scientific articles. Peer review is essential to the journal. My special thanks to Mark Newman, M.D., for contributing his splendid photographs for our cover; and to Gwynneth Wilson and the auxiliary for our special feature on History of Medicine in Alaska.

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Letters to the Editor

On Unification

Dear Editor:

I noted in the most recent AMA News that your state medical society is contemplating unification with the AMA. The same article announced that Delaware had just become the 6th state to unify. Virginia had been the 5th to do so slightly over a year ago. As a past president (1979) of the Medical Society of Virginia (MSV), may I urge that you carefully review the recent experience of the MSV with this controversial move prior to such a commitment?

Virginia unified at our annual meeting in November 1985. This was accomplished by a vote of our House of Delegates without a membership referendum as clearly provided in our bylaws for "matters of sufficient importance"; this question (unification) clearly qualified for that category.

Nothing has so divided our Society since mandatory CME in 1976 and ramifications of the most serious significance are appearing. Our A.M.A. delegation made up almost exclusively of past-presidents, and thereby exerting enormous influence and pressure on Councilors and Delegates, was intent on the election of a Virginian as president-elect of the A.M.A. This was accomplished but at a great price. At the time of adoption the A.M.A. presidential election was just six months away, and they did not politically dare to risk the possible deleterious effects of unification rejection. A more democratic and appropriate membership referendum was thus denied.

Many serious spin-offs have occurred. I will list a few:

1. Marked bitterness within the Society.
2. The loss of more than a thousand dues-paying members (15-20% of our membership), many of whom feel that they were denied due democratic process.
3. Marked loss of revenue because of #2. This has necessitated severe budgetary restrictions, adversely affecting some long-term and/or important programs and Society functions.
4. The levying of a $105 dues increase for 1987.
5. The request that more than 700 dues exempt members resume payment of dues. Many of this group are ill, elderly and impoverished.

Our experience obviously has been dismal. Your membership would be wise to study it deliberately and in great depth before adoption.

Charles E. Davis, Jr., M.D.
Past President
Medical Society of Virginia

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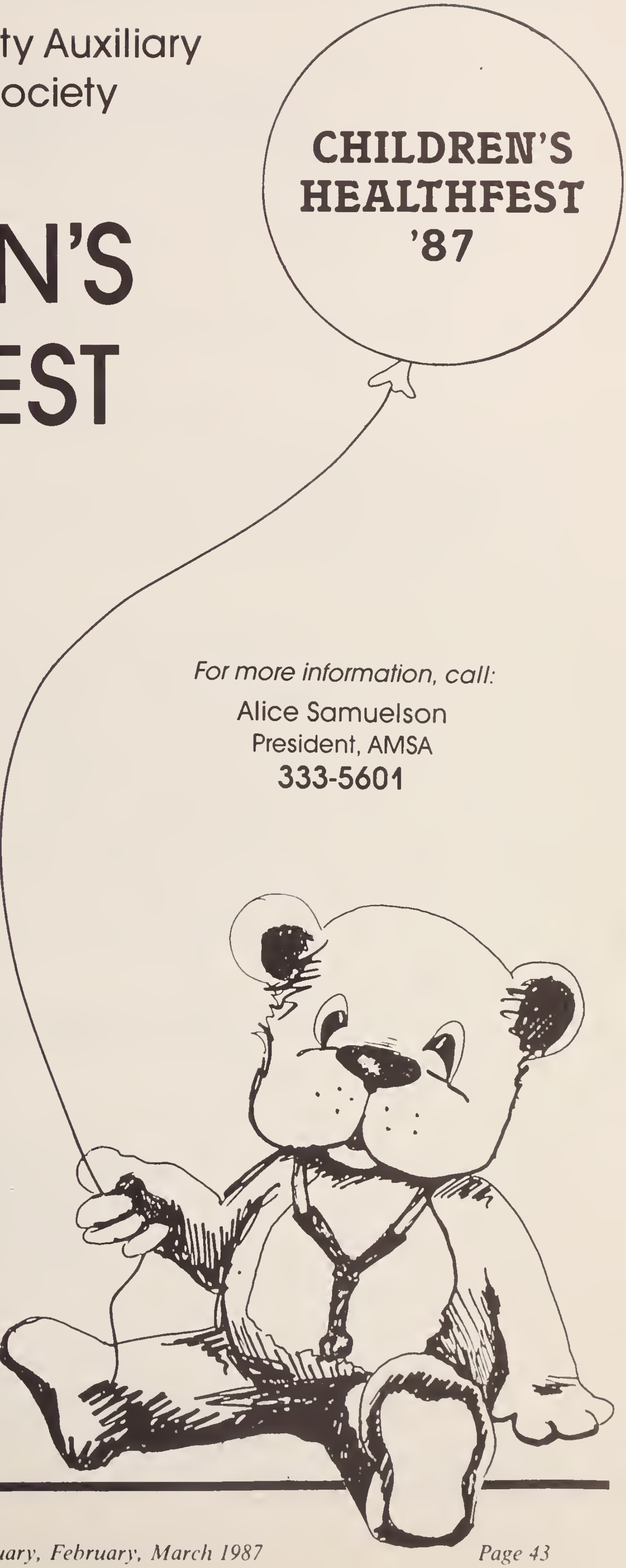

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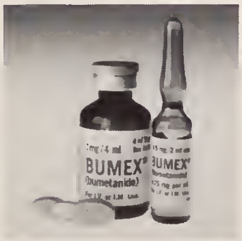

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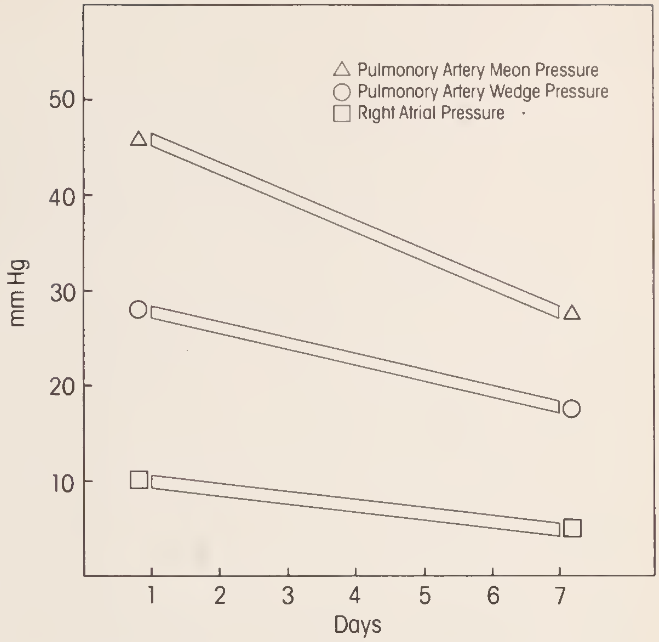

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ALASKA MEDICINE

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AMERICAN SOCIETY FOR CIRCUMPOLAR HEALTH

Volume 29, Number 2 April, May, June 1987

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NEW LOW DOSE
INDERAL LA 60 mg
(PROPRANOLOL HCl) LONG ACTING CAPSULES

Please see next page for brief summary of prescribing information

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride INDERAL LA is available as 60 mg 80 mg 120 mg and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL the chronotropic inotropic and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (60 80 120 and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings however such as hypertension or angina where there is little correlation between plasma levels and clinical effect INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis especially for treatment of exertional or other stress-induced angina, palpitations and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock 2) sinus bradycardia and greater than first-degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

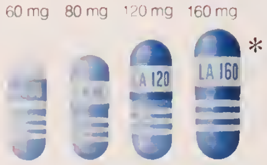
WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema) —PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case, this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. GENERAL Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lea return of increased intraocular pressure

CLINICAL LABORATORY TESTS Elevated blood urea levels in patients with severe disease elevated serum transaminase alkaline phosphatase lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as pine should be closely observed if INDERAL is administered The added catechola blocking action may produce an excessive reduction of resting sympathetic nervous a which may result in hypotension marked bradycardia vertigo syncopal attacks or ortho hypotension

Caution should be exercised when patients receiving a beta blocker are administer calcium-channel-blocking drug especially intravenous verapamil for both agents ma press myocardial contractility or atrioventricular conduction On rare occasions the con tant intravenous use of a beta blocker and verapamil has resulted in serious adverse reac especially in patients with severe cardiomyopathy congestive heart failure or recent m dial infarction

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol

Ethanol slows the rate of absorption of propranolol

Phenytoin phenobarbitone, and *rifampin* accelerate propranolol clearance

Chlorpromazine when used concomitantly with propranolol results in increased p levels of both drugs

Antipyrine and *lidocaine* have reduced clearance when used concomitantly propranolol

Thyroxine may result in a lower than expected T_3 concentration when used concom with propranolol

Cimetidine decreases the hepatic metabolism of propranolol delaying eliminatio increasing blood levels

Theophylline clearance is reduced when used concomitantly with propranolol

CARCINOGENESIS MUTAGENESIS, IMPAIRMENT OF FERTILITY Long-term stud animals have been conducted to evaluate toxic effects and carcinogenic potential month studies in both rats and mice, employing doses up to 150 mg kg day there w evidence of significant drug-induced toxicity There were no drug-related tumorigenic e at any of the dosage levels Reproductive studies in animals did not show any impairm fertility that was attributable to the drug

PREGNANCY Pregnancy Category C INDERAL has been shown to be embryoto animal studies at doses about 10 times greater than the maximum recommended human

There are no adequate and well-controlled studies in pregnant women INDERAL sho used during pregnancy only if the potential benefit justifies the potential risk to the fetus

NURSING MOTHERS INDERAL is excreted in human milk Caution should be exer when INDERAL (propranolol HCl) is administered to a nursing woman

PEDIATRIC USE Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have required the withdrawal of therapy

Cardiovascular Bradycardia, congestive heart failure; intensification of AV block hyp sion paresthesia of hands, thrombocytopenic purpura arterial insufficiency, usually Raynaud type

Central Nervous System Light-headedness, mental depression manifested by inso lassitude weakness fatigue reversible mental depression progressing to catatonia disturbances, hallucinations vivid dreams, an acute reversible syndrome characteriz disorientation for time and place short memory loss, emotional lability, sl clouded sensorium, and decreased p mance on neuropsychometrics For imme formulations, fatigue, lethargy and dreams appear dose related

Gastrointestinal Nausea, vomiting, e tric distress, abdominal cramping dia constipation mesenteric arterial throm ischemic colitis

Allergic Pharyngitis and agranulocy erythematous rash, fever combined wit ing and sore throat, laryngospasm and re tory distress

Respiratory Bronchospasm

Hematologic Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic pu

Auto-Immune In extremely rare instances, systemic lupus erythematosus has reported

Miscellaneous Alopecia LE-like reactions, psoriasiform rashes, dry eyes, male impo and Peyronie's disease have been reported rarely Oculomucocutaneous reactions inv the skin serous membranes and conjunctivae reported for a beta blocker (practolol) ha been associated with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochlori sustained-release capsule for administration once daily If patients are switched from IND Tablets to INDERAL LA Capsules, care should be taken to assure that the desired thera effect is maintained INDERAL LA should not be considered a simple mg-for-mg substi INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitratio be necessary especially to maintain effectiveness at the end of the 24-hour dosing inte

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is INDERAL LA once daily whether used alone or added to a diuretic The dosage m increased to 120 mg once daily or higher until adequate blood-pressure control is ach The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage mg may be required The time needed for full hypertensive response to a given dos variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDER once daily, dosage should be gradually increased at three- to seven-day intervals until o response is obtained Although individual patients may respond at any dosage lev average optimal dosage appears to be 160 mg once daily In angina pectoris, the val safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued reduce dosage gradually over a period of a few wee WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDER once daily The usual effective dose range is 160-240 mg once daily The dosage m increased gradually to achieve optimal migraine prophylaxis If a satisfactory response obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy be discontinued It may be advisable to withdraw the drug gradually over a period of weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group ar limited to permit adequate directions for use

*The appearance of these capsules is a registered trademark of Ayerst Laboratories

REFERENCES:

1. INDERAL LA National Compliance Evaluation Program Data on file Ayerst Laborat
2. Ravid M, Lang R, Jutrin I The relative antihypertensive potency of propranolol oxpr atenolol, and metoprolol given once daily *Arch Intern Med* 1985, 145 1321-1323

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Ayerst

AYERST LABORATORIES
New York, NY 10017

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CONFIRMED

CONFIRMED BY CLINICAL EVIDENCE

ZANTAC® 150 h.s.

ranitidine HCl/Glaxo 150 mg tablets

EFFECTIVE MAINTENANCE THERAPY

for healed duodenal ulcer patients

See last page for references and
Brief Summary of Product Information.

Glaxo/ROCHE

CONFIRMED

In two randomized, double-blind, and well-controlled clinical trials, ZANTAC 150 mg h.s. significantly superior to cimetidine 400 mg h.s. for maintenance therapy in healed duodenal ulcers.

Percent of patients with observed duodenal ulcer recurrence

		0-4 months	0-8 months	0-12 months	No. patients
USA[1]	ranitidine 150 mg h.s.	9%	14%*	16%†	60
	cimetidine 400 mg h.s.	23%	34%	43%	66
UK, Ireland, Australia[2]	ranitidine 150 mg h.s.	8%‡	14%‡	23%‡	243
	cimetidine 400 mg h.s.	21%	34%	37%	241

*p=0.02
†p=0.01
‡p≤0.004
%=life-table estimates
All patients were permitted prn antacids for relief of pain.

These two trials used the currently recommended dosing regimen of cimetidine (400 mg h.s.) and ranitidine (150 mg h.s.). A comparison of other dosing regimens has not been studied.

The studied dosing regimens are not equivalent with respect to the degree and duration of acid suppression or suppression of nocturnal acid.

The superiority of ranitidine over cimetidine in these trials indicates that the dosing regimen currently recommended for cimetidine is less likely to be as successful in maintenance therapy.

Convenient once-a-night dose with a low incidence of side effects[3]

Headache, sometimes severe, seems to be related to ranitidine administration. Other side effects have been reported; for a complete listing, see the **ADVERSE REACTIONS** section in the Brief Summary.

No significant interference with the hepatic cytochrome P-450 enzyme system at recommended doses

ZANTAC 150 mg has no significant drug interactions with theophylline, phenytoin, or warfarin. The bioavailability of certain medications whose absorption is dependent on a low gastric pH may be altered when ZANTAC or other medications that decrease gastric acidity are administered.

Zantac® 150
ranitidine HCl/Glaxo 150 mg tablets

One tablet at bedtime
for maintenance

See next page for references and
Brief Summary of Product Information.

Glaxo/ROCHE®

CONFIRMED

Zantac® 150

ranitidine HCl/Glaxo *150 mg tablets*

One tablet at bedtime for maintenance therapy in healed duodenal ulcer patients

References:

1. Silvis SE, Griffin J, Hardin R, et al: Final report on the United States multicenter trial comparing ranitidine to cimetidine as maintenance therapy following healing of duodenal ulcer. *J Clin Gastroenterol* 1985;7(6):482-487.
2. Gough KR, Korman MG, Bardhan KD, et al Ranitidine and cimetidine in prevention of duodenal ulcer relapse: A double-blind, randomised, multicentre, comparative trial. *Lancet* 1984;ii:659-662.
3. Data available on request, Glaxo Inc.

ZANTAC® 150 Tablets
(ranitidine hydrochloride)
ZANTAC® 300 Tablets
(ranitidine hydrochloride)

BRIEF SUMMARY OF PRODUCT INFORMATION

The following is a brief summary only. Before prescribing, see complete prescribing information in ZANTAC® product labeling.

INDICATIONS AND USAGE: ZANTAC® is indicated in:

1. Short-term treatment of **active duodenal ulcer.** Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD).** Symptomatic relief commonly occurs within one or two weeks after starting therapy and is maintained throughout a six-week course of therapy.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

CONTRAINDICATIONS: ZANTAC® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS: Symptomatic response to ZANTAC® therapy does not preclude the presence of gastric malignancy.

Since ZANTAC is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see **DOSAGE AND ADMINISTRATION**). Caution should be observed in patients with hepatic dysfunction since ZANTAC is metabolized in the liver.

False-positive tests for urine protein with Multistix® may occur during ZANTAC therapy, and therefore testing with sulfosalicylic acid is recommended.

Although recommended doses of ZANTAC do not inhibit the action of cytochrome P-450 enzymes in the liver, there have been isolated reports of drug interactions which suggest that ZANTAC may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Lack of experience to date precludes recommending ZANTAC for use in children or pregnant patients. Since ZANTAC is secreted in human milk, caution should be exercised when administered to a nursing mother.

ADVERSE REACTIONS: Headache, sometimes severe, seems to be related to ZANTAC® administration. Constipation, diarrhea, nausea/vomiting, and abdominal discomfort/pain have been reported. There have been rare reports of malaise, dizziness, somnolence, insomnia, vertigo, tachycardia, bradycardia, premature ventricular beats, and arthralgias. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients.

In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid IV for seven days, and in 4 of 24 subjects receiving 50 mg qid for five days. With oral administration there have been occasional reports of reversible hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice.

There have been rare reports of reversible leukopenia, granulocytopenia, thrombocytopenia, and pancytopenia.

Although controlled studies have shown no antiandrogenic activity, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving ZANTAC, but the incidence did not differ from that in the general population.

Incidents of rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia, have been reported, as well as rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia) and small increases in serum creatinine.

OVERDOSAGE: Information concerning possible overdosage and its treatment appears in the full prescribing information.

DOSAGE AND ADMINISTRATION Active Duodenal Ulcer: The current recommended adult oral dosage is 150 mg twice daily. An alternate dosage of 300 mg once daily at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated.

Maintenance Therapy: The current recommended adult oral dosage is 150 mg at bedtime.

Pathological Hypersecretory Conditions (such as Zollinger-Ellison Syndrome): The current recommended adult oral dosage is 150 mg twice a day. In some patients it may be necessary to administer ZANTAC 150-mg doses more frequently. Doses should be adjusted to individual patient needs, and should continue as long as clinically indicated. Doses up to 6 g/day have been employed in patients with severe disease.

Benign Gastric Ulcer: The current recommended adult oral dosage is 150 mg twice a day.

GERD: The current recommended adult oral dosage is 150 mg twice a day.

Dosage Adjustment for Patients with Impaired Renal Function: On the basis of experience with a group of subjects with severely impaired renal function treated with ZANTAC, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

HOW SUPPLIED: ZANTAC® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) and unit dose packs of 100 tablets (NDC 0173-0393-47).

ZANTAC® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 tablets (NDC 0173-0344-42) and unit dose packs of 100 tablets (NDC 0173-0344-47).

Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.

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Glaxo

Glaxo Inc.
Research Triangle Park, NC 27709

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"Living in the city is lonely enough... with herpes it's like solitary confinement."
ZOVIRAX®
(acyclovir)
CAPSULES
Prevent genital herpes recurrences month after month with daily therapy.
(In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.)
Please see last page of this advertisement for brief summary of prescribing information.

ZOVIRAX®
(acyclovir)
CAPSULES

Help free your patients from recurrences.

Daily therapy

Coping with genital herpes is rarely easy. For some, the worst part is the pain and discomfort of frequent attacks —month after month, year after year. For others, the emotional burden presents a more difficult problem, leading to social isolation, anxiety, and diminished self-esteem.

Prevent or reduce recurrences

Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

Generally well tolerated

Daily therapy with ZOVIRA CAPSULES is generally wel tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/ vomiting, vertigo, and arthralgia.

The physical and emotiona difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episode as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRA CAPSULES.

Please see brief summary of prescribing information on next pa

Prevent recurrences month after month*

ZOVIRAX®

(acyclovir)

CAPSULES

Brief Summary

INDICATIONS AND USAGE: Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psychosocial difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

First Episodes (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

Recurrent Episodes:

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

CONTRAINDICATIONS: Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

WARNINGS: Zovirax Capsules are intended for oral ingestion only.

PRECAUTIONS: General: Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

Drug Interactions: Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the F_1 generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

Pregnancy: ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies,. the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS — Short-Term Administration: The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

Long-Term Administration: The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

Chronic suppressive therapy for recurrent disease: One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

Intermittent Therapy: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

Patients With Acute or Chronic Renal Impairment: One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance ≤ 10 ml/min/1.73/m².

HOW SUPPLIED: Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200"- Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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*In controlled studies , recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

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ALASKA MEDICINE

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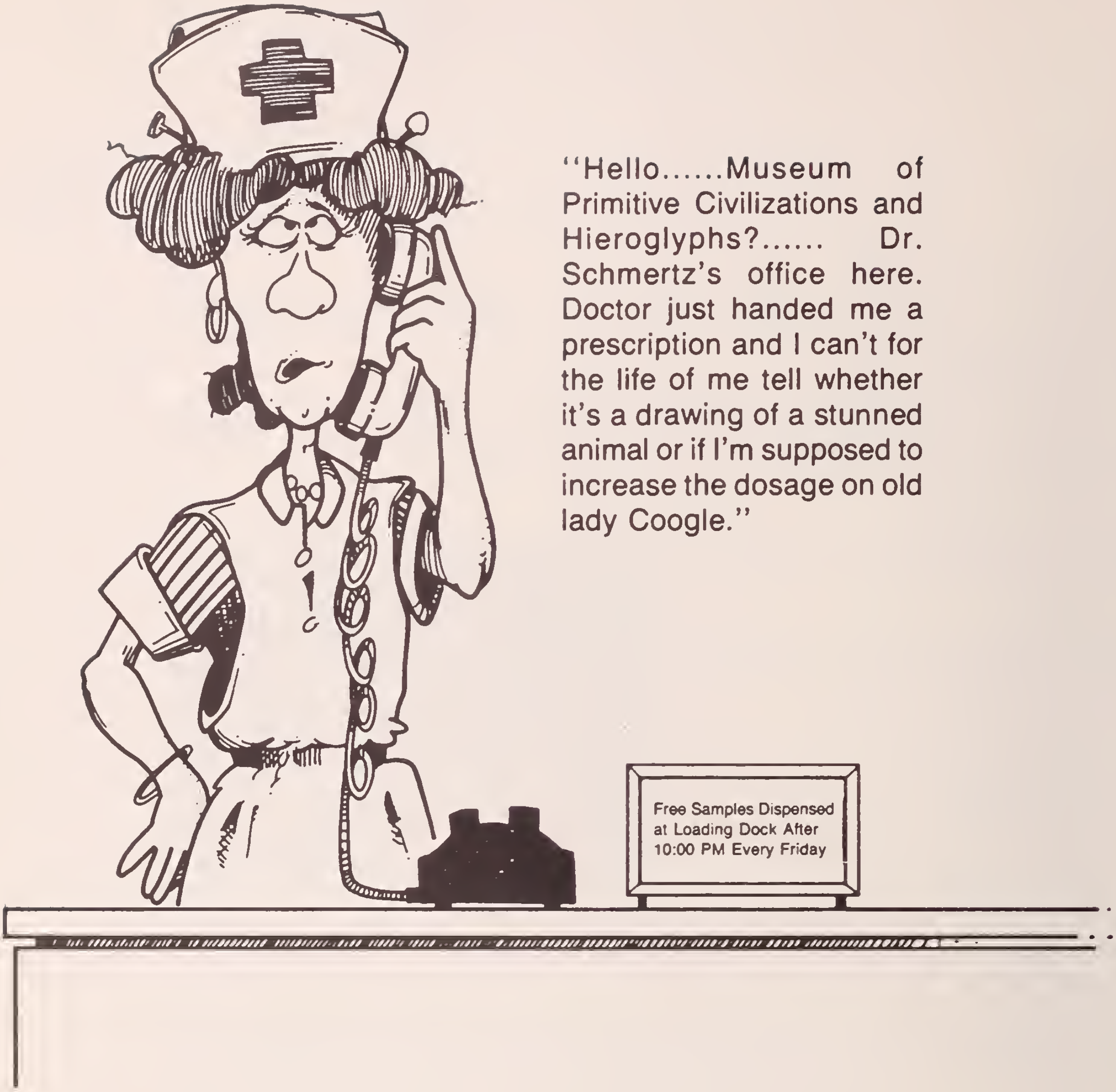
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Twenty Ways to Stay Alive Longer in Anchorage

by Rodman Wilson, M.D.[1]

In 1985, 794 persons died in Anchorage, Alaska. Cardiovascular disease accounted for 27.6% of deaths, malignant neoplasm 24.9%, violence and untoward effects 24.9%, and all other causes 22.6%.

Violence and untoward effects in this context includes deaths from motor vehicle, aircraft and bicycle accidents, falls, fire, drowning and boating accidents, suicide, homicide, alcoholism, and inadvertent overdosage of drugs.

The proportion of deaths in the nation in 1984 due to violence, thus defined, was only 8.4%. This was one-third the proportion in Anchorage in 1985 dying violently.

Midyear 1985 population of Anchorage was estimated to be 230,434. The crude death rate for 794 deaths was 3.45 deaths per 1,000 population. This was far lower than the preliminary crude death rate for 1984 for the United States of 8.57.

However, when the death rate for Anchorage is adjusted for age, the rate rises to 9.32. Despite the youth of our populace, death is more frequent by 9% then in the nation as a whole, where average age is considerably higher. Excess mortality in Anchorage is strongly suspected of being due to violence.

In an attempt to publicize this, I sent a list of 20 admonitions to the public to the press, using common language as much as possible. Only portions of the document were published by newspapers. Play was more jocular than solemn. Distribution was fairly wide throughout Alaska.

The homily is published here in its full form. Salient references have been added to support assertions not self-evident.

TWENTY WAYS TO STAY ALIVE LONGER IN ANCHORAGE

1. STAY AWAY FROM VIOLENT PEOPLE

   They kill and maim (1–4)

2. AVOID LOADED GUNS

   More than 30,000 persons die annually from gunfire in the USA (5–7)

3. PLACE SMOKE ALARMS IN YOUR HOME AND DO NOT SMOKE IN BED

   Home fires can be avoided (8)

   Drinking and then smoking is particularly dangerous (9, 10)

4. WEAR LIFE PRESERVERS WHEN BOATING

   It is difficult to survive cold Alaskan waters, even if you can swim

5. DRIVE CAUTIOUSLY AND COURTEOUSLY

   Truculent drivers cause many injuries and deaths even when not drunk

6. WEAR AUTO SEAT BELTS; PUT YOUR SMALL CHILDREN INTO CHILD CAR SEATS

   This will reduce injuries and deaths by haif (11)

7. DO NOT DRINK AND THEN DRIVE, FLY, BOAT, SWIM, BIKE, OR WALK IN THE STREET

   Too many drivers drink (5, 12)

   Too many pilots drink (13)

   One half of pedestrians killed by vehicles have been drinking (5)

   Beer and boating do not mix (14, 15)

8. LIMIT ALCOHOL TO ONE OR TWO DRINKS DAILY

   A little alcohol is not harmful unless it is followed by hazardous activity or unless it leads to heavier drinking (16)

   Pregnant women should not drink at all (17)

9. DO NOT USE ILLEGAL DRUGS

   Cocaine kills (18)
   Many other illicit drugs dull awareness and thereby cause accidents (19, 20)

10. THINK SAFETY ON THE JOB

    Wear helmets and safety shoes on construction and other hazardous jobs (21)

---

[1] Deputy Director, Department of Health & Human Services, Municipality of Anchorage.

Be careful not to fall (5, 22)

Do not let your boss assign you to a dangerous job without training and protection

Ask about toxic materials at work, but remember that most of the time cigarette smoke is the most toxic chemical at the workplace (23, 24)

11. LIVE MONOGAMOUSLY

This is a way to avoid serious sexually transmitted diseases and entangling alliances

Married people live longer (25)

12. DO NOT SMOKE AND AVOID THE SMOKE OF OTHERS

This greatly reduces the chance that you will get cancer of the mouth, larynx, lung, esophagus, stomach, pancreas, kidney, bladder, and cervix (26–29)

Also you will not likely ever get chronic bronchitis or emphysema (27, 30, 31)

Your children will grow taller, have less asthma and lung infection, and will be at less risk of getting many types of cancer when they grow up (32–34)

13. REDUCE THE AMOUNT OF FAT IN YOUR DIET BY HALF

Fat in the blood accelerates hardening of the arteries; this leads to coronary heart attacks and other serious blood vessel disorders such as, stroke, aneurysm, and gangrene (35–38)

Persons who consume a lot of fat have more cancer of the colon and breast (39, 40)

14. IF YOU HAVE HIGH BLOOD PRESSURE, GET IT TREATED

Controlling high blood pressure prevents stroke and heart failure (41, 42)

15. IF YOU HAVE DIABETES, GET IT TREATED

This will make you feel better and will prevent or delay life-shortening complications (43)

16. IF YOU ARE DEPRESSED, SEEK HELP

Depression is eminently treatable (44)

Suicide need not happen

17. IF YOU ARE A WOMAN, GET A BREAST EXAMINATION AND A PAP TEST PERIODICALLY

Many breast cancers found early can be cured (45)

No one need die from cancer of the cervix (46)

18. IF YOU ARE PREGNANT,

SEE YOUR PHYSICIAN EARLY

Prenatal care helps prevent low birth weight that kills or maims many babies (47–49)

DO NOT SMOKE

Smoking causes babies to be small (50)

DO NOT DRINK

Alcohol damages babies (17, 51)

DO NOT USE ILLEGAL DRUGS

They harm babies (52, 53)

19. KEEP VACCINATIONS UP-TO-DATE

This applies to adults as well as children (54)

Influenza and pneumococcal vaccines are particularly important for elderly and debilitated individuals (55, 56)

Vaccines to prevent certain cancers are at hand (57); others will come

20. GET A PET

Some claim that people who own pets are healthier and happier; proof is lacking (58–62)

ACKNOWLEDGMENT

I thank Rose Courtright for assistance in preparing these admonitions.

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Hot Tub Folliculitis

by Sue Anne Jenkerson, M.S.N., R.N.C.
John Middaugh, M.D.[1]

ABSTRACT

An outbreak of pseudomonas folliculitis due to *Pseudomonas aeruginosa* was documented among all 11 members of a softball team who used an Alaskan spa. Significant findings were painful, indurated, apocrine axillary glands in four (36%) of seven individuals examined and inflammation of Montgomery's follicules in one individual. Patients presenting with discrete papulopustular rash, axillary tenderness, or inflammed Montgomery's follicules and a recent history of hot tub, whirlpool, or swimming pool use should be suspected of having pseudomonas folliculitis.

INTRODUCTION

Although outbreaks of pseudomonas folliculitis associated with hot tubs, whirlpools, and swimming pools have frequently been described (1-4), clinical diagnosis of a single case can be difficult. Since 1983, three outbreaks of pseudomonas folliculitis in Alaska involving a private hot tub party (5), a wrestling team using a whirlpool (6), and most recently, a softball team visiting an Alaskan spa (7) have been investigated. This paper summarizes the softball team outbreak, presenting clinical data from seven cases in an effort to help physicians and other health care providers to diagnose pseudomonas folliculitis and to recognize potential outbreaks.

METHODS

Between August 20 and 22, 1986, we interviewed all 14 team members and examined seven members with rash illness. On August 22, skin cultures from five individuals were obtained with cotton swabs and transported in appropriate media within four hours to the Southcentral Laboratory, Section of Laboratories, Division of Public Health for culture. We obtained informed consent to photograph the rashes of three individuals. We mailed a questionnaire to 49 spa members who reportedly used the implicated spa on the same day as the softball team. A follow-up survey of non-responders was not done. Sanitarians from the Alaska Department of Environmental Conservation and the local health agency inspected the spa and took samples of pool and hot tub surfaces and water for culture. All environmental cultures were also done at the Southcentral Laboratory.

RESULTS

On August 16, 1986, after participating in a post-season softball tournament, members of one team relaxed at an Alaskan Health Spa. Within 24 hours, three of the 14 team members developed a rash. As team members talked to one another, they discovered other members with similar complaints. Several members had sought medical care and were treated for staphylococcal skin infections. We interviewed all 14 team members. Of the 14 team members, all 11 who used the spa developed a similar rash illness, attack rate = 100%. The three team members who did not enter the spa reported no illness. On August 22, 1986 we examined seven members of the softball team; all had a rash.

Skin lesions were discrete, erythematous, and papulopustular (Figure 1 and 2) and were scattered over thighs, buttocks, upper arms, chests, and breasts (Table 1). One individual had painful inflammation of the Montgomery's follicles of the right breast (Figures 3 and 4). A significant finding among team members was swollen, indurated, painful axillary glands in four (36%) individuals. Other reported symptoms were fever (27%), muscle aches (27%), malaise (27%), pruritis (18%), and chills (9%). Two team members complained of urinary tract infection (UTI) symptoms after visiting the spa. Both were taking antibiotics when examined. We did not obtain urine cultures. A third individual was taking Amoxicillin for a UTI when she used the spa facilities. No one had otitis externa or toe web infections.

Onset of rash and other related symptoms ranged from 24 to 90 hours with a mean onset of 50 hours. Time spent in the tub ranged from 5 to 35 minutes with a mean duration of 20 minutes. All 11 members who entered the spa used the hot tub; nine individuals reported alternating between tub and pool several times. Members reported that the hot tub water was "super hot" but cloudy. All members wore one-piece bathing suits.

Of 49 spa members who used the spa the same day as the softball team, 20 (41%) responded to our questionnaire. Of these 20, three women reported rash illness

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[1] Epidemiology Office, Division of Public Health, Department of Health and Social Services, State of Alaska.

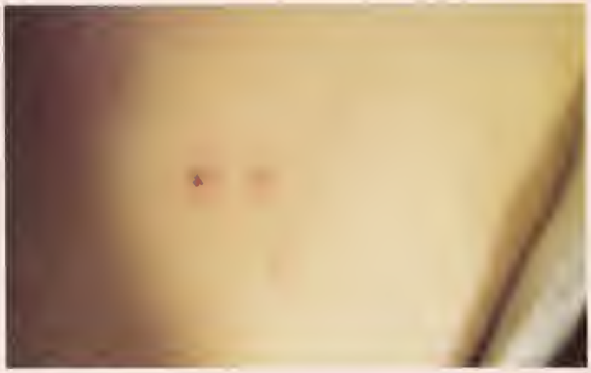

Figure 1

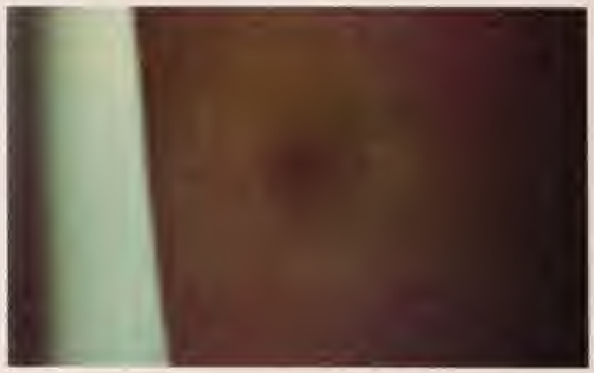

Figure 2

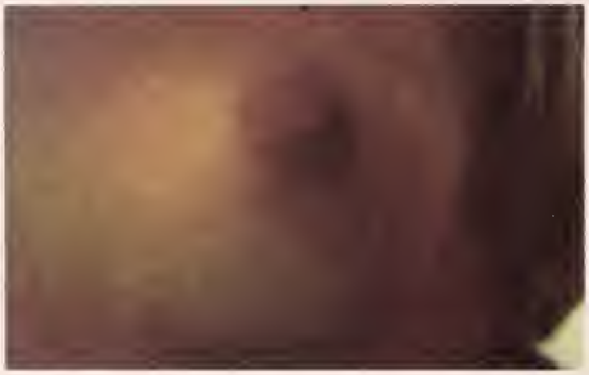

Figure 3

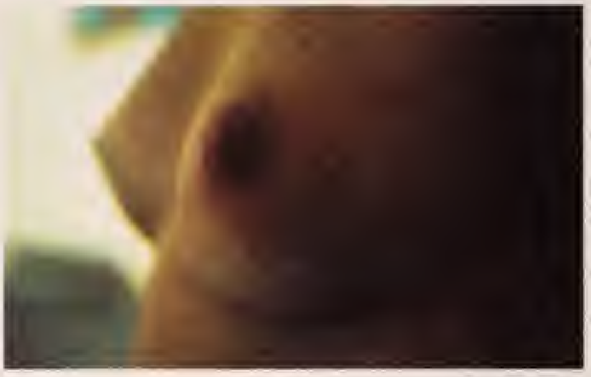

Figure 4

after using spa facilities. One of these individuals reported painful swelling of her breasts, and a second individual reported a painful red lump in her armpit. One respondent also reported that 2 women in a water aerobics class in May developed rashes after using the pool.

Investigation of the facility revealed that the hot tub had not been cleaned for six days prior to the outbreak, although the tub had a large user rate (50–75/day). In addition, the owner reused disposable filters after backwashing them. Although chlorination and pH levels that had been recorded by the owner appeared adequate, often only one reading per day was recorded.

Specimens from one individual's lesions, the hot tub sidewall, filter and water, and the swimming pool water produced *Pseudomonas aeruginosa,* the well-documented culprit of hot tub folliculitis.

The pool and hot tub were closed on August 22, 1986 until proper disinfection and cleaning procedures were performed, including discarding all used filters. After cleaning, lab tests of the pool and hot tub were negative for pseudomonas. The spa was allowed to reopen.

DISCUSSION

This outbreak typifies hot tub pseudomonas folliculitis. Outbreaks often remain undiagnosed unless several members of a family, sports team, or other group of individuals notice a similar rash after common exposure (4). Once an outbreak is identified, high attack rates often are found. In this outbreak, exposed team members suffered a 100% attack rate. The high attack rates leave few or no non-ill controls making it hard to evaluate risk factors (4).

The rash associated with hot tub folliculitis is not unique in its appearance and is often misdiagnosed as contact dermatitis, scabies, staphylococal infection, drug allergy, insect bites, or even herpes zoster (2, 4). Lesions in many different stages are often present simultaneously. Each lesion progresses from a discrete follicular, pruritic papule to an erythematous papulopustule within 24 to 28 hours (2). Papules range from two to ten millimeters in diameter but usually have only a pinpoint pustule in the center. Papulopustules heal spontaneously either by draining or by regressing within two to five days, leaving fine desquamation and red-brown macules (2, 4).

A characteristic feature of this rash is its distribution. Pseudomonas has a predilection for apocrine sweat glands. Lesions are most often observed on the skin of the buttocks, hips, thighs, and lateral trunk (4). Painful inflammation of Montgomery's follicles, modified apocrine glands located in the breast areola, is often observed (4). Tender, swollen, red axillary glands may be the presenting symptom to the health care provider (2). The rash does not occur on palms, soles, or mucus membranes. In addition to the rash, many infected individuals complain of headaches, malaise, fatigue, low grade fever, abdominal cramping, nausea, vomiting, sore throat, and sore eyes (1, 2, 4). Although not seen in this outbreak, otitis externa often occurs with pseudomonas folliculitis (2). Other signs which may occur are green nail syndrome, a distinct discoloration under the nail with green pigment, and toe web infections (2).

Superhydration of the stratum corneum, such as occurs from prolonged sitting in a hot tub, leads to high surface concentrations of pseudomonas that predispose the skin to invasion (8). The high temperature associated with hot tubs promotes sweating which enhances penetration of the skin by the organism. Skin entry may occur via minor abrasions sustained when the body is rubbed against the walls of the tub (1).

Chronic skin disorders such as acne may predispose to or increase the severity of the pseudomonas infection. Compromised immune status has not been identified as a predisposing condition of pseudomonas folliculitis. In fact, most cases occur in exceptionally athletic and healthy individuals (4). The overall morbidity associated with outbreaks appears relatively minor. Most lesions resolve without medication in 7 to 10 days.

In addition to skin lesions, hot tub pseudomonas has also been associated with urinary tract infections (UTI) (9), pulmonary infections (10), and possible systemic illness resulting in peripheral inflammatory nodules (5, 11). Only a few patients become systemically ill (11). Although two individuals in this outbreak reported UTI symptoms after using the spa, no urine cultures were obtained prior to starting antibiotics. Because we did not collect urine specimens, we do not know if these symptoms were due to pseudomonas-caused UTIs.

The ability of *P. aeruginosa* to withstand relatively high temperatures (up to 41.4° C) and even high chlorine levels (total residual chlorine concentration of 3.0 mg/L) makes it difficult to eradicate the organisms from hot tubs and other public bathing places. A heavy bather load and turbulent water produce difficulties in maintaining satisfactory levels of chlorine. With heavy bather use, desquamated skin in the water provides a rich organic nutrient source for pseudomonas (1). Wooden tubs are particularly a problem. It is believed that once pseudomonas contaminates a wooden hot tub, eradication is impossible. Pseudomonas has been found in pores and cracks of the wood itself. Oregon has banned commercial use of wood-lined hot tubs (12).

Culture surveys of swimming pools show that *P. aeruginosa* is frequently present in low numbers and persists despite proper chlorination. The organism multiplies rapidly when the free chlorine level drops below 0.5 mg/L or the water pH is greater than 7.8. High temperatures and water agitation in hot tubs cause an increased loss of chlorine into the air. Proper maintenance of filters, continuous water filtration, automatic chlorination to maintain a free chlorine level of between 2 to 5 ppm,, frequent monitoring of the disinfectant level, maintenance of water at a pH of 7.2 to 7.8, and the frequent changing of water, especially during heavy use, are some helpful measures that should be applied to all public and private bath places to prevent this disease (13).

Patients presenting with papulopustular rashes, painful indurated apocrine axillary glands, and a history of hot tub, whirlpool, or swimming pool use should be suspected of pseudomonas folliculitis. Inflammed Montgomery's follicles in a patient who has recently used a hot tub should strongly suggest the diagnosis of pseudomonas. A thorough skin exam should be performed looking for typical lesions which should be cultured for the organism. No treatment is required for uncomplicated infections. Systemic symptoms occur infrequently, but may require parenteral administration of aminoglycoside antibiotics. Diagnosed or suspected cases should be reported promptly so that epidemiologic investigation can determine if a common source outbreak has occurred.

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Table 1. Patient data on individuals with skin lesions after visiting health spa, August 1986.

Patient	Sex/Age	Site of Lesions	Other Symptoms	Lab
1	F/34	Buttocks, axilla		
2	F/34	Upper arm		
3	F/29	Trunk, Axilla, Breasts	Nausea	
4	F/29	Trunk	Malaise, fever muscle ache, Possible UTI	Pseudomonas aeruginosa
5	F/28	Buttocks, Arm, Axilla		
6	F/16	Trunk, Leg, Buttocks	Painful swollen Montgomery's follicle, pruritis	
7	F/35	Trunk, Neck, Arms, Axilla	Possible UTI, malaise, chills, severe pruritis	

The Physician's Role in Physical Activity, Physical Fitness and Health

By Jay Caldwell, M.D.[1]

The U.S. Department of Health and Human Services has established as a national goal the participation by 60% of adults aged 18-65 in REGULAR and VIGOROUS physical activity by 1990 (1). USA Today reports that already we are a nation in which 100 million people swim, 75 million bicycle, 75 million "do exercises," and 35 million jog (2). And we haven't even factored avid walkers into this compilation.

Furthermore, the 1979 National Survey of Personal Health Practices (3) indicated that 78% of adults are active "often" in physical activities. I can name another ten surveys, with a cumulative sample size well in excess of 100,000, in which the percent of active adults ranges between 49 and 68 (4, 5, 6). This sounds great. We must already be there.

On the other hand, there is a gloomy side to this. These surveys, which seem to have such a grand sweep, are prisoners of their methodology (7). Rarely is the frequency and vigor of participation accurately tallied; definitions of physical activity and exercise are fuzzy as well as inconsistent, making study-to-study comparisons difficult at best (8).

A night out at the bowling alley or an occasional backyard round of volleyball, while providing affirmative answers in some of these surveys, is certainly not what the folks over at DHHS had in mind when they drew up that goal for us.

In surveys that do address these points the results are chilling. The 1982 CDC State Behavioral Risk Factor Survey (9), with 22,200 telephone interviews logged, reported that only 21% of respondents indicated sufficient fitness activity (3+ kcal/kg/day in a single exercise activity).

A face-to-face and telephone survey (N = 1510) privately commissioned by Perrier, the bottled water group, and conducted by Louis Harris and Associates in 1978 reported that only 15% achieved an exercise volume of 1500 kcal/week (5).

Still, questions about sample size and reliability, geographical and age bias, self-selection of respondents, and errors inherent in non-respondency remain. In 1984 the Simmons Market Research Bureau undertook, in conjunction with the Department of Sports Management and Communication at the University of Michigan, to put these concerns to rest in a survey of 19,000 adults (10). They looked not just simply at involvement, but also at the intensity and long-term frequency of participation. The results are discouraging.

They found that although 53% took part ("at least once") in activities of which the DHHS would approve, only 18.6% did so at least weekly. Within the total group of 19 activities, a "fitness cluster" consisting of swimming, bicycling, jogging, and weight lifting was identified; at least weekly participation was indicated by no more than 14% of the sample. USA Today's 100 million swimmers dwindles rapidly to 4.9 million adults who swim at least 60 days throughout the year.

There is no doubt that exercise is good for us. It has beneficial effects on both our physical (11) and mental health (12), even if we may not understand the mechanisms. These benefits strongly outweigh the toll of all the injuries and side effects which accrue from a program of regular physical fitness.

Although I have never seen a study which has addressed the degree of physician involvement in the domain of patients' physical activity, there is a recent report from the Michigan Department of Health and the CDC (13) that indicates that in the years 1980 and 1983, only 44% of smokers stated that they had EVER been advised by their doctors to quit. This is appalling when you consider that a 1979 Surgeon General's report has estimated that 10-25% of smokers so counselled will indeed cease smoking on that basis alone (13). I suspect there is even less discussion of exercise in doctors' offices.

It is not easy to distill down the volumes of research done on this subject, but it is possible to prioritize what matters about exercise (14). The most crucial element is that something gets done. Then comes how much, followed by what. Bringing up the rear is how hard you do it, which paradoxically seems to be the point that gets the most publicity and turns off the majority of those who almost, but don't quite, get around to making a start. We have this inherent sense that if we don't do it right, when why bother.

It isn't even that hard to achieve a cardiovascular training effect, if it is that on which you are insistent. At the Department of Exercise Science at the University of Massachusetts, a series of studies was undertaken to determine the efficacy of walking in elevating the pulse into the training range (15).

The Massachusetts workers found that 80+% of all women were able to walk fast enough to achieve a training effect (70% of their maximal heart rate), and although the success rate was lower among men, it, not surprisingly, increased with the age. Only men in the 30–39 age span and the fittest group of 40–49 year-old men, would find walking not to be a sufficient training stress.

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Average walking paces, in minutes per mile for age groups, necessary for women to achieve a training heart rate were: 13:38 minutes (30–39 years), 14:17 minutes (40–49 years), 15:00 minutes (50–59 years), and 15:23 minutes (60–69 years). For men in the same age brackets, the necessary paces worked out to be 12:46, 12:30, 12:46, and 13:57 minutes, respectively. Bear in mind that at a mile pace better than 12:00–12:30 minutes per mile (4.8–5.0 mph), jogging becomes more efficient, mechanically, than walking. Studies that have attempted to quantitate the amount of activity necessary to achieve a health benefit, be it reduced coronary disease or overall mortality, are in surprising agreement (16, 17). The requisite minimal daily kilocalorie expenditure for leisure activity is in the range of 250–300 (3–4 kcal/kg/day). When work activities have been considered in terms of reduced heart disease risk, there is agreement that about twice that number of kilocalories is required.

None of this is to say that such immutable principles of physiology as the necessity for overloading a system to induce a training effect can be violated. There is, as I have heard said, no free lunch. Rather, the point is simply that one has to be doing something before you can do it right. As physicians, our emphasis should be on the encouragement of getting started (Table 1) (16). It will take months to addict a newcomer to the pleasures of being active. Once the habit is formed, the questions of how hard, how much, and what mixture can be addressed.

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Note—Culture and susceptibility tests should be initiated prior to and during therapy. Renal function studies should be performed when indicated.

Contraindication: Keflet is contraindicated in patients with known allergy to the cephalosporin group of antibiotics

Warnings: BEFORE CEPHALEXIN THERAPY IS INSTITUTED CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS AND PENICILLIN. CEPHALOSPORIN C DERIVATIVES SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS

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There is some clinical and laboratory evidence of partial cross-allergenicity of the penicillins and the cephalosporins. Patients have been reported to have had severe reactions (including anaphylaxis) to both drugs.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics cautiously. No exception should be made with regard to Keflet

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should be ruled out

Usage in Pregnancy—Safety of this product for use during pregnancy has not been established.

Precautions: *General*—Patients should be followed carefully so that any side effects or unusual manifestations of drug idiosyncrasy may be detected. If an allergic reaction to Keflet occurs, the drug should be discontinued and the patient treated with the usual agents (eg, epinephrine or other pressor amines, antihistamines, or corticosteroids)

Prolonged use of Keflet may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Keflet should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

Indicated surgical procedures should be performed in conjunction with antibiotic therapy.

As a result of administration of Keflet, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly)

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—The daily oral administration of cephalexin to rats in doses of 250 or 500 mg/kg prior to and during pregnancy, or to rats and mice during the period of organogenesis only, had no adverse effect on fertility, fetal viability, fetal weight, or litter size. Note that the safety of cephalexin during pregnancy in humans has not been established

Cephalexin showed no enhanced toxicity in weanling and newborn rats as compared with adult animals. Nevertheless, because the studies in humans cannot rule out the possibility of harm, Keflet should be used during pregnancy only if clearly needed

Nursing Mothers— The excretion of cephalexin in the milk increased up to 4 hours after a 500-mg dose; the drug reached a maximum level of 4μg/mL then decreased gradually, and had disappeared 8 hours after administration. Caution should be exercised when Keflet is administered to a nursing woman

Adverse Reactions: *Gastrointestinal*—Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely. The most frequent side effect has been diarrhea. It was very rarely severe enough to warrant cessation of therapy. Dyspepsia and abdominal pain have also occurred. As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely

Hypersensitivity—Allergic reactions in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens Johnson Syndrome, or toxic epidermal necrolysis have been observed. These reactions usually subsided upon discontinuation of the drug. Anaphylaxis has also been reported

Other reactions have included genital and anal pruritus, genital moniliasis, vaginitis and vaginal discharge, dizziness, fatigue, and headache. Eosinophilia, neutropenia, thrombocytopenia, and slight elevations in SGOT and SGPT have been reported

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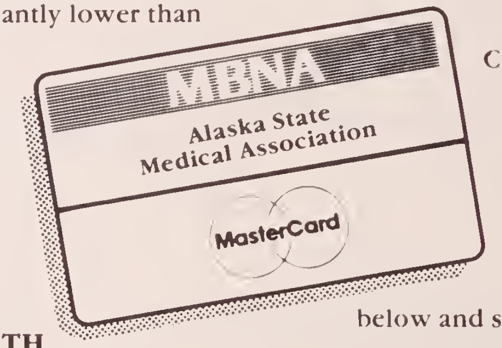

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ntraindications: Concomitant use with other potassium-sparing agents ch as spironolactone or amiloride. Further use in anuria, progressive nal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum tassium. Hypersensitivity to either component or other sulfonamide-rived drugs.

arnings: **Do not use potassium supplements, dietary or otherwise, less hypokalemia develops or dietary intake of potassium is markedly paired.** If supplementary potassium is needed, potassium tablets ould not be used. Hyperkalemia can occur, and has been associated th cardiac irregularities. It is more likely in the severely ill, with urine lume less than one liter/day, the elderly and diabetics with suspected confirmed renal insufficiency. Periodically, serum K^+ levels should be termined. If hyperkalemia develops, substitute a thiazide alone, restrict $^+$ intake. **Associated widened QRS complex or arrhythmia requires ompt additional therapy.** Thiazides cross the placental barrier and pear in cord blood. Use in pregnancy requires weighing anticipated nefits against possible hazards, including fetal or neonatal jaundice, rombocytopenia, other adverse reactions seen in adults. Thiazides pear and triamterene may appear in breast milk. If their use is essential, e patient should stop nursing. Adequate information on use in children not available. Sensitivity reactions may occur in patients with or with-t a history of allergy or bronchial asthma. Possible exacerbation or tivation of systemic lupus erythematosus has been reported with iazide diuretics.

recautions: The bioavailability of the hydrochlorothiazide component of yazide' is about 50% of the bioavailability of the single entity. eoretically, a patient transferred from the single entities of triamterene d hydrochlorothiazide may show an increase in blood pressure or fluid tention. Similarly, it is also possible that the lesser hydrochlorothiazide oavailability could lead to increased serum potassium levels. However, tensive clinical experience with 'Dyazide' suggests that these conditions ve not been commonly observed in clinical practice. Angiotensin-nverting enzyme (ACE) inhibitors can elevate serum potassium; use ith caution with 'Dyazide'. Do periodic serum electrolyte determinations articularly important in patients vomiting excessively or receiving renteral fluids, and during concurrent use with amphotericin B or rticosteroids or corticotropin [ACTH]). Periodic BUN and serum eatinine determinations should be made, especially in the elderly, abetics or those with suspected or confirmed renal insufficiency. umulative effects of the drug may develop in patients with impaired renal nction. Thiazides should be used with caution in patients with impaired patic function. They can precipitate coma in patients with severe liver sease. Observe regularly for possible blood dyscrasias, liver damage, her idiosyncratic reactions. Blood dyscrasias have been reported in tients receiving triamterene, and leukopenia, thrombocytopenia, ranulocytosis, and aplastic and hemolytic anemia have been reported ith thiazides. Thiazides may cause manifestation of latent diabetes ellitus. The effects of oral anticoagulants may be decreased when ed concurrently with hydrochlorothiazide; dosage adjustments may be cessary. Clinically insignificant reductions in arterial responsiveness norepinephrine have been reported. Thiazides have also been shown to crease the paralyzing effect of nondepolarizing muscle relaxants such tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic ood studies in cirrhotics with splenomegaly. Antihypertensive effects ay be enhanced in post-sympathectomy patients. Use cautiously in rgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' ould be used with caution in patients with histories of stone formation. few occurrences of acute renal failure have been reported in patients 'Dyazide' when treated with indomethacin. Therefore, caution is dvised in administering nonsteroidal anti-inflammatory agents with yazide'. The following may occur: transient elevated BUN or creatinine both, hyperglycemia and glycosuria (diabetic insulin requirements may e altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), ecreasing alkali reserve with possible metabolic acidosis. 'Dyazide' terferes with fluorescent measurement of quinidine. Hypokalemia is ncommon with 'Dyazide', but should it develop, corrective measures hould be taken such as potassium supplementation or increased dietary take of potassium-rich foods. Corrective measures should be instituted autiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated erum potassium. Chloride deficit may occur as well as dilutional yponatremia. Concurrent use with chlorpropamide may increase the risk severe hyponatremia. Serum PBI levels may decrease without signs thyroid disturbance. Calcium excretion is decreased by thiazides. yazide' should be withdrawn before conducting tests for parathyroid nction. Thiazides may add to or potentiate the action of other anti-ypertensive drugs. Diuretics reduce renal clearance of lithium and crease the risk of lithium toxicity.

dverse Reactions: Muscle cramps, weakness, dizziness, headache, ry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other ermatological conditions; nausea and vomiting, diarrhea, constipation, ther gastrointestinal disturbances; postural hypotension (may be ggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, aresthesias, icterus, pancreatitis, xanthopsia and respiratory distress ncluding pneumonitis and pulmonary edema, transient blurred vision, ialadenitis, and vertigo have occurred with thiazides alone. Triamterene as been found in renal stones in association with other usual calculus omponents. Rare incidents of acute interstitial nephritis have been eported. Impotence has been reported in a few patients on 'Dyazide', lthough a causal relationship has not been established.

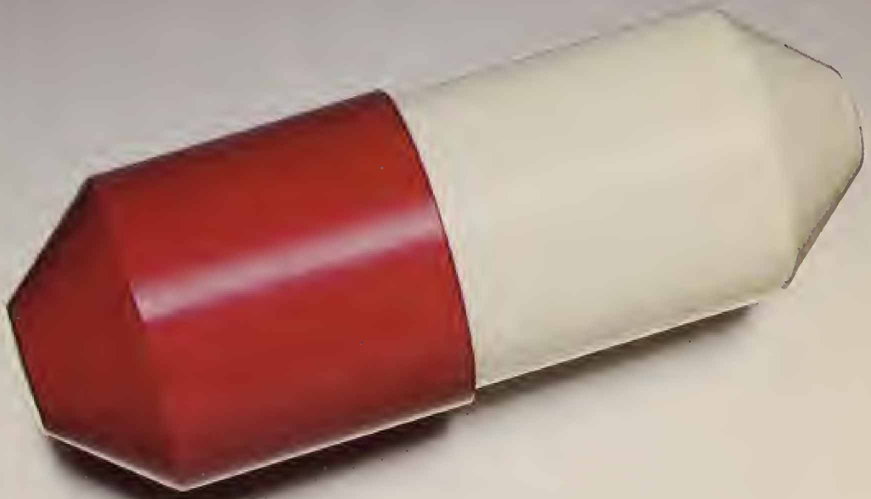
upplied: 'Dyazide' is supplied as a red and white capsule, in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for nstitutional use only); in Patient-Pak™ unit-of-use bottles of 100.

RS-DZ L42

In Hypertension*... When You Need to Conserve K^+

Remember the Unique Red and White Capsule: Your Assurance of SK&F Quality

Serum K^+ and BUN should be checked periodically (see Warnings and Precautions).



Potassium-Sparing

DYAZIDE®

25 mg Hydrochlorothiazide/**50 mg** Triamterene/SKF

Over 20 Years of Confidence

The unique
red and white
Dyazide® capsule:
Your assurance of
SK&F quality.

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Effective control time and time again[1]

Effective control of fasting and postprandial glucose—patient after patient, meal after meal, year after year.

Insulin when it's needed

Insulin levels are rapidly elevated in response to a meal, then return promptly to basal levels after the meal challenge subsides.

Timed to minimize risks

Rapidly metabolized and excreted, with an excellent safety profile.[1] As with all sulfonylureas, hypoglycemia may occur.

In concert with diet in non-insulin-dependent diabetes mellitus

Glucotrol®
(glipizide) 5-mg and 10-mg Scored Tablets

SYNCHRONIZED SULFONYLUREA THERAPY

Please see brief summary of Glucotrol® (glipizide) prescribing information on next page.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

Reference:

1. Sachs R, Frank M, Fishman SK Overview of clinical experience with glipizide In *Glipizide A Worldwide Review* Princeton, NJ, Excerpta Medica, 1984, pp 163-172

GLUCOTROL® (glipizide) Tablets

Brief Summary of Prescribing Information

INDICATIONS AND USAGE: GLUCOTROL is indicated as an adjunct to diet for the control of hyperglycemia in patients with non-insulin-dependent diabetes mellitus (NIDDM, type II) after an adequate trial of dietary therapy has proved unsatisfactory

CONTRAINDICATIONS: GLUCOTROL is contraindicated in patients with known hypersensitivity to the drug or with diabetic ketoacidosis, with or without coma, which should be treated with insulin

SPECIAL WARNING ON INCREASED RISK OF CARDIOVASCULAR MORTALITY: The administration of oral hypoglycemic drugs has been reported to be associated with increased cardiovascular mortality as compared to treatment with diet alone or diet plus insulin. This warning is based on the study conducted by the University Group Diabetes Program (UGDP), a long-term prospective clinical trial designed to evaluate the effectiveness of glucose-lowering drugs in preventing or delaying vascular complications in patients with non-insulin-dependent diabetes. The study involved 823 patients who were randomly assigned to one of four treatment groups (*Diabetes*, 19, supp. 2:747-830, 1970).

UGDP reported that patients treated for 5 to 8 years with diet plus a fixed dose of tolbutamide (1.5 grams per day) had a rate of cardiovascular mortality approximately 2-1/2 times that of patients treated with diet alone. A significant increase in total mortality was not observed, but the use of tolbutamide was discontinued based on the increase in cardiovascular mortality, thus limiting the opportunity for the study to show an increase in overall mortality. Despite controversy regarding the interpretation of these results, the findings of the UGDP study provide an adequate basis for this warning. The patient should be informed of the potential risks and advantages of GLUCOTROL and of alternative modes of therapy.

Although only one drug in the sulfonylurea class (tolbutamide) was included in this study, it is prudent from a safety standpoint to consider that this warning may also apply to other oral hypoglycemic drugs in this class, in view of their close similarities in mode of action and chemical structure.

PRECAUTIONS: **Renal and Hepatic Disease**: The metabolism and excretion of GLUCOTROL may be slowed in patients with impaired renal and/or hepatic function Hypoglycemia may be prolonged in such patients should it occur

Hypoglycemia: All sulfonylureas are capable of producing severe hypoglycemia Proper patient selection, dosage, and instructions are important to avoid hypoglycemia Renal or hepatic insufficiency may increase the risk of hypoglycemic reactions Elderly, debilitated or malnourished patients and those with adrenal or pituitary insufficiency are particularly susceptible to the hypoglycemic action of glucose-lowering drugs Hypoglycemia may be difficult to recognize in the elderly or people taking beta-adrenergic blocking drugs Hypoglycemia is more likely to occur when caloric intake is deficient, after severe or prolonged exercise, when alcohol is ingested, or when more than one glucose-lowering drug is used

Loss of Control of Blood Glucose: A loss of control may occur in diabetic patients exposed to stress such as fever, trauma, infection or surgery It may then be necessary to discontinue GLUCOTROL and administer insulin

Laboratory Tests: Blood and urine glucose should be monitored periodically Measurement of glycosylated hemoglobin may be useful

Information for Patients: Patients should be informed of the potential risks and advantages of GLUCOTROL, of alternative modes of therapy, as well as the importance of adhering to dietary instructions, of a regular exercise program, and of regular testing of urine and/or blood glucose The risks of hypoglycemia, its symptoms and treatment, and conditions that predispose to its development should be explained to patients and responsible family members Primary and secondary failure should also be explained

Drug Interactions: The hypoglycemic action of sulfonylureas may be potentiated by certain drugs including nonsteroidal anti-inflammatory agents and other drugs that are highly protein bound, salicylates, sulfonamides, chloramphenicol, probenecid, coumarins, monoamine oxidase inhibitors, and beta adrenergic blocking agents *In vitro* studies indicate that GLUCOTROL binds differently than tolbutamide and does not interact with salicylate or dicumarol However, caution must be exercised in extrapolating these findings to a clinical situation Certain drugs tend to produce hyperglycemia and may lead to loss of control, including the thiazides and other diuretics, corticosteroids, phenothiazines, thyroid products, estrogens, oral contraceptives, phenytoin, nicotinic acid, sympathomimetics, calcium channel blocking drugs, and isoniazid A potential interaction between oral miconazole and oral hypoglycemic agents leading to severe hypoglycemia has been reported Whether this interaction also occurs with the intravenous, topical, or vaginal preparations of miconazole is not known

Carcinogenesis, Mutagenesis, Impairment of Fertility: A 20-month study in rats and an 18-month study in mice at doses up to 75 times the maximum human dose revealed no evidence of drug-related carcinogenicity Bacterial and *in vivo* mutagenicity tests were uniformly negative Studies in rats of both sexes at doses up to 75 times the human dose showed no effects on fertility

Pregnancy: Pregnancy Category C· GLUCOTROL (glipizide) was found to be mildly fetotoxic in rat reproductive studies at all dose levels (5-50 mg/kg) This fetotoxicity has been similarly noted with other sulfonylureas, such as tolbutamide and tolazamide The effect is perinatal and believed to be directly related to the pharmacologic (hypoglycemic) action of GLUCOTROL In studies in rats and rabbits no teratogenic effects were found There are no adequate and well controlled studies in pregnant women. GLUCOTROL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Because recent information suggests that abnormal blood glucose levels during pregnancy are associated with a higher incidence of congenital abnormalities, many experts recommend that insulin be used during pregnancy to maintain blood glucose levels as close to normal as possible

Nonteratogenic Effects: Prolonged severe hypoglycemia has been reported in neonates born to mothers who were receiving a sulfonylurea drug at the time of delivery This has been reported more frequently with the use of agents with prolonged half-lives GLUCOTROL should be discontinued at least one month before the expected delivery date

Nursing Mothers: Since some sulfonylurea drugs are known to be excreted in human milk insulin therapy should be considered if nursing is to be continued

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS: In controlled studies, the frequency of serious adverse reactions reported was very low Of 702 patients, 11 8% reported adverse reactions and in only 1 5% was GLUCOTROL discontinued

Hypoglycemia: See PRECAUTIONS and OVERDOSAGE sections

Gastrointestinal: Gastrointestinal disturbances, the most common, were reported with the following approximate incidence nausea and diarrhea, one in 70, constipation and gastralgia, one in 100 They appear to be dose-related and may disappear on division or reduction of dosage Cholestatic jaundice may occur rarely with sulfonylureas GLUCOTROL should be discontinued if this occurs

Dermatologic: Allergic skin reactions including erythema, morbilliform or maculopapular eruptions, urticaria pruritus, and eczema have been reported in about one in 70 patients These may be transient and may disappear despite continued use of GLUCOTROL, if skin reactions persist, the drug should be discontinued Porphyria cutanea tarda and photosensitivity reactions have been reported with sulfonylureas

Hematologic: Leukopenia, agranulocytosis, thrombocytopenia, hemolytic anemia, aplastic anemia, and pancytopenia have been reported with sulfonylureas

Metabolic: Hepatic porphyria and disulfiram-like alcohol reactions have been reported with sulfonylureas Clinical experience to date has shown that GLUCOTROL has an extremely low incidence of disulfiram-like reactions

Endocrine Reactions: Cases of hyponatremia and the syndrome of inappropriate antidiuretic hormone (SIADH) secretion have been reported with this and other sulfonylureas

Miscellaneous: Dizziness, drowsiness, and headache have been reported in about one in fifty patients treated with GLUCOTROL They are usually transient and seldom require discontinuance of therapy

OVERDOSAGE: Overdosage of sulfonylureas including GLUCOTROL can produce hypoglycemia If hypoglycemic coma is diagnosed or suspected, the patient should be given a rapid intravenous injection of concentrated (50%) glucose solution This should be followed by a continuous infusion of a more dilute (10%) glucose solution at a rate that will maintain the blood glucose at a level above 100 mg/dL Patients should be closely monitored for a minimum of 24 to 48 hours since hypoglycemia may recur after apparent clinical recovery Clearance of GLUCOTROL from plasma would be prolonged in persons with liver disease Because of the extensive protein binding of GLUCOTROL (glipizide), dialysis is unlikely to be of benefit.

DOSAGE AND ADMINISTRATION: There is no fixed dosage regimen for the management of diabetes mellitus with GLUCOTROL, in general, it should be given approximately 30 minutes before a meal to achieve the greatest reduction in postprandial hyperglycemia

Initial Dose: The recommended starting dose is 5 mg before breakfast Geriatric patients or those with liver disease may be started on 2 5 mg Dosage adjustments should ordinarily be in increments of 2 5-5 mg, as determined by blood glucose response At least several days should elapse between titration steps

Maximum Dose: The maximum recommended total daily dose is 40 mg

Maintenance· Some patients may be effectively controlled on a once-a-day regimen, while others show better response with divided dosing Total daily doses above 15 mg should ordinarily be divided

HOW SUPPLIED: GLUCOTROL is available as white, dye-free, scored diamond-shaped tablets imprinted as follows 5 mg tablet—Pfizer 411 (NDC 5 mg 0049-4110-66) Bottles of 100, 10 mg tablet—Pfizer 412 (NDC 10 mg 0049-4120-65) Bottles of 100

CAUTION Federal law prohibits dispensing without prescription

More detailed professional information available on request.

ROERIG Pfizer A division of Pfizer Pharmaceuticals
New York, New York 10017

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"YOU'D DIE IF YOU HAD MY JOB."
YOU COULD LEARN A LOT FROM A DUMMY. BUCKLE YOUR SAFETY BELT.

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Motrin® 800 TABLETS mg
ibuprofen

Extra strength
Convenience
Economy

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100 A Century of Caring
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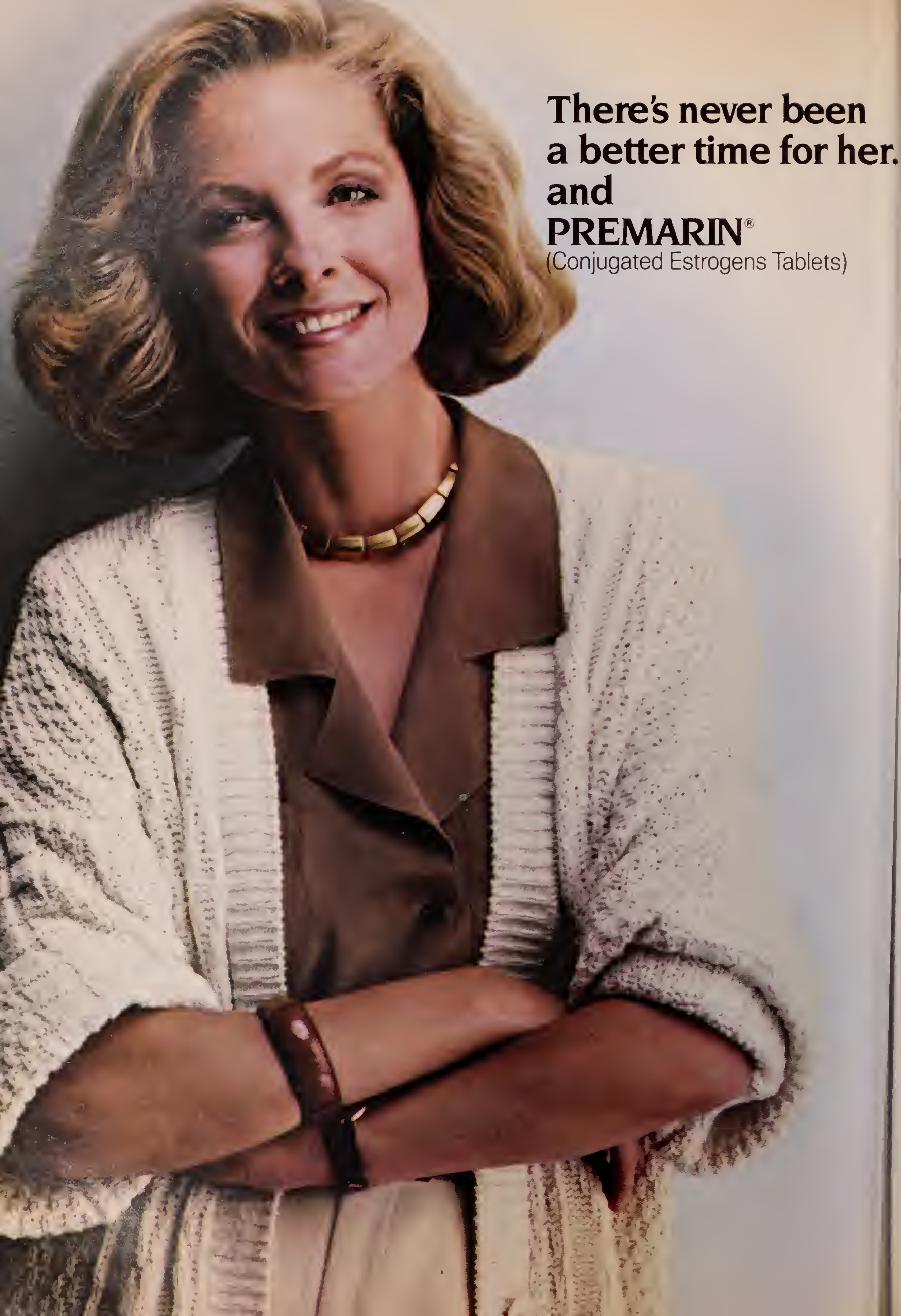
There's never been a better time for her.
and
PREMARIN®
(Conjugated Estrogens Tablets)

Now the evidence looks better than ever

Significantly reduced risk of endometrial hyperplasia

Endometrial hyperplasia was significantly reduced when progestin was added to PREMARIN therapy for more than ten days a month.[1-4] The risk of endometrial hyperplasia may also be reduced through cyclic administration of unopposed, low-dose PREMARIN.

Effect on lipids—an important feature

PREMARIN used alone does not adversely affect lipid levels. In fact, a clinical study has shown a significant increase in HDL cholesterol—from 49.7 mg/dL to 56.4 mg/dL—and decrease in LDL cholesterol—from 165.1 mg/dL to 138.1 mg/dL—after one year of therapy with PREMARIN, 0.625 mg.[5]

Low-dose control of menopausal symptoms*

PREMARIN effectively relieves vasomotor symptoms, such as hot flashes. When estrogen deficiency is limited to atrophic vaginitis, PREMARIN® (conjugated estrogens) Vaginal Cream restores the vaginal environment to its premenopausal state.

The most widely used, most extensively studied estrogen worldwide.

PREMARIN®

(Conjugated Estrogens Tablets)

Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

For moderate-to-severe vasomotor symptoms

PREMARIN®
(Conjugated Estrogens Tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®
(Conjugated Estrogens)

Vaginal Cream
0.625mg/g

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base

> 1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.
>
> Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses.
>
> 2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.
>
> The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4.7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0 3 mg, 0 625 mg, 0 9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass)**. Atrophic vaginitis. Kraurosis vulvae Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae. PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS.) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions: 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2. Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (See Boxed Warning). 4 Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

WARNINGS: Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility. There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease, or abnormal mammograms A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrog
per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase
risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis When doses of this size
used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderne
abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estrog
containing oral contraceptives Increased blood pressure may occur with use of estrogens in the menopause
blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observe
patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be caref
observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to
initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic orga
and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer t
one year without another physical examination being performed Conditions influenced by fluid retention suc
asthma. epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients
develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleedi
mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the
of endometrial hyperplasia in some patients Oral contraceptives appear to be associated with an increa
incidence of mental depression Patients with a history of depression should be carefully observed. Preexist
uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estro
therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen,
medication should be discontinued while the cause is investigated Estrogens should be used with care in patie
with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, o
young patients in whom bone growth is not complete. If concomitant progestin therapy is used, potential r
may include adverse effects on carbohydrate and lipid metabolism

The following changes may be expected with larger doses of estrogen.

a Increased sulfobromophthalein retention

b Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3; increased
epinephrine-induced platelet aggregability.

c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone,
measured by PBI, T4 by column, or T4 by radioimmunoassay Free T3 resin uptake is decreased, reflecting
elevated TBG; free T4 concentration is unaltered

d Impaired glucose tolerance

e Decreased pregnanediol excretion

f. Reduced response to metyrapone test.

g Reduced serum folate concentration.

h. Increased serum triglyceride and phospholipid concentration. As a general principle, the administratio
any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in hu
milk

ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including oral contracepti
breakthrough bleeding, spotting, change in menstrual flow; dysmenorrhea; premenstrual-like syndro
amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, chang
cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secre
(of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma w
may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; los
scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migra
dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance; aggra
tion of porphyria; edema; changes in libido.

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females.

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1. *Given cyclically for short-term use only.* For treatment of moderate to severe *vasomotor* symptoms, atro
vaginitis, or kraurosis vulvae associated with the menopause (0 3 to 1.25 mg or more daily). The lowest dose
will control symptoms should be chosen and medication should be discontinued as promptly as possi
Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or ta
medication should be made at three- to six-month intervals.

2 *Given cyclically:* Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically. Ad
upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level
will provide effective control. Osteoporosis —0.625 mg daily. Administration should be cyclic (eg, three we
on and one week off).

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measu
taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only. For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinue
promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer
appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recur
abnormal vaginal bleeding

References:

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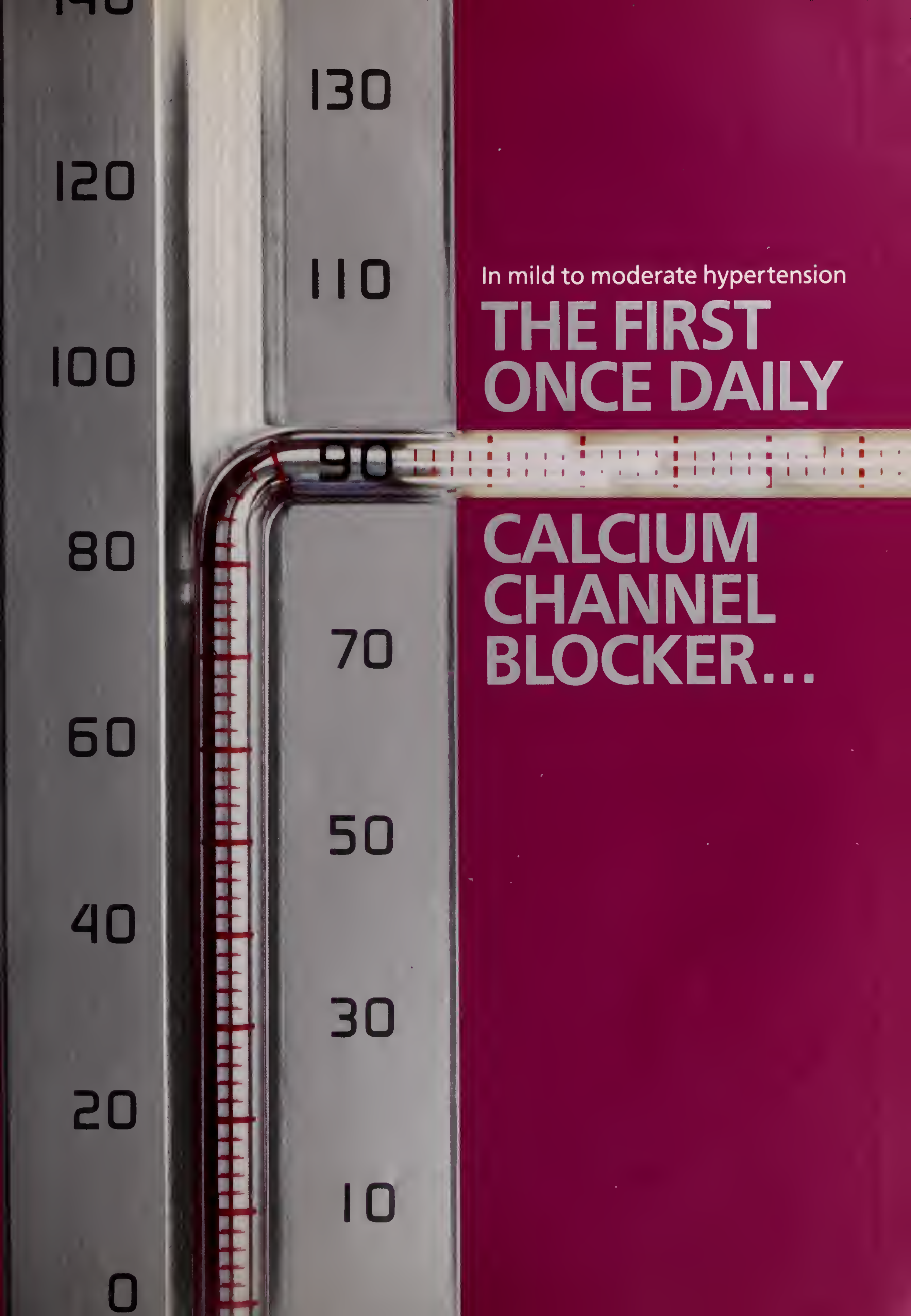
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In mild to moderate hypertension
THE FIRST ONCE DAILY
CALCIUM CHANNEL BLOCKER...

NEW
ONCE DAILY

ISOPTIN SR*

(verapamil HCl/Knoll)

240 mg scored, sustained-release tablets

JAMES B.
38, black male, heavy smoker. Prescribed a diuretic by another physician last year for hypertension.

YOUR CONCERNS
Presents with "smoker's cough." Workup reveals a BP of 150/107.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN* (verapamil HCl/Knoll) because...
— Black hypertensives often have low plasma renin activity and generally do not respond favorably to beta blockers.
— Beta blockers may increase the likelihood of bronchospasm.

ALICE W.
65, diabetic, overweight. Her BP has elevated to 190/98.

YOUR CONCERNS
She's on daily insulin.

A LOGICAL CHOICE FOR CONTROL OF HER BP
ISOPTIN* (verapamil HCl/Knoll) because...
— Unlike most beta blockers and diuretics, ISOPTIN has no adverse effects on serum glucose levels.
— Unlike most beta blockers, ISOPTIN does not mask the symptoms of hypoglycemia.

THOMAS G.
70, asthmatic. In the past, BP adequately controlled with 25 mg hydrochlorothiazide daily.

YOUR CONCERNS
Today patient presents with symptoms of gout. Workup reveals high uric acid level, low serum potassium, and BP elevated to 180/98.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN* (verapamil HCl/Knoll) because...
— Unlike diuretics, ISOPTIN will not decrease serum potassium levels or elevate uric acid levels.
— Unlike beta blockers, ISOPTIN can be used safely in asthma and COPD patients.

JOHN K.
42, Annual physical uncovered diastolic BP of 102... confirmed on three successive office visits. Unresponsive to nonpharmacologic intervention.

YOUR CONCERNS
Salesman, spends many hours of his working day in car... total cholesterol level 300, HDL 35.

A LOGICAL CHOICE FOR CONTROL OF HIS BP
ISOPTIN* (verapamil HCl/Knoll) because...
— Unlike diuretics, ISOPTIN does not cause urinary urgency.
— Unlike either beta blockers or diuretics, ISOPTIN will not adversely affect his already seriously compromised lipid profile.
— Unlike with propranolol, fatigue and impotence are rarely reported.

Antihypertensive therapy you and your patients can live with

Knoll Pharmaceuticals
A Unit of BASF K&F Corporation
Whippany, New Jersey 07981

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Please see adjacent page for brief summary.

In mild to moderate hypertension

Brief Summary

THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER

ISOPTIN® SR
(verapamil HCl/Knoll)
240 mg scored, sustained-release tablets

CONTRAINDICATIONS: 1) Severe left ventricular dysfunction (see WARNINGS), 2) Hypotension (less than 90 mmHg systolic pressure) or cardiogenic shock, 3) Sick sinus syndrome or 2nd or 3rd degree AV block (except in patients with a functioning artificial ventricular pacemaker).

WARNINGS: Heart Failure: ISOPTIN should be avoided in patients with severe left ventricular dysfunction (see DRUG INTERACTIONS). Patients with milder ventricular dysfunction should, if possible, be controlled before verapamil treatment. Hypotension: ISOPTIN (verapamil HCl) may produce occasional symptomatic hypotension. Elevated Liver Enzymes: Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Periodic monitoring of liver function in patients receiving verapamil is therefore prudent. Accessory Bypass Tract (Wolff-Parkinson-White): Patients with paroxysmal and/or chronic atrial flutter or atrial fibrillation and a coexisting accessory AV pathway have developed increased antegrade conduction across the accessory pathway producing a very rapid ventricular response or ventricular fibrillation after receiving intravenous verapamil. While this has not been reported with oral verapamil, it should be considered a potential risk Treatment is usually D.C.-cardioversion. Atrioventricular Block: The effect of verapamil on AV conduction and the SA node may cause asymptomatic 1st degree AV block and transient bradycardia. Higher degrees of AV block, while infrequent (0.8%), may require a reduction in dosage or, in rare instances, discontinuation of verapamil HCl. Patients with Hypertrophic Cardiomyopathy (IHSS): Although verapamil has been used in the therapy of patients with IHSS, severe cardiovascular decompensation and death have been noted in this patient population.

PRECAUTIONS: Impaired Hepatic or Renal Function: Verapamil is highly metabolized by the liver with about 70% of an administered dose excreted in the urine. In patients with impaired hepatic or renal function verapamil should be administered cautiously and the patients monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacological effects (see OVERDOSAGE).

Drug Interactions: Beta Blockers: Concomitant use of ISOPTIN and oral beta-adrenergic blocking agents may be beneficial in certain patients with chronic stable angina or hypertension, but available information is not sufficient to predict with confidence the effects of concurrent treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities. Digitalis: Clinical use of verapamil in digitalized patients has shown the combination to be well tolerated if digoxin doses are properly adjusted. However, chronic verapamil treatment increases serum digoxin levels by 50 to 75% during the first week of therapy and this can result in digitalis toxicity. Upon discontinuation of ISOPTIN (verapamil HCl), the patient should be reassessed to avoid underdigitalization. Antihypertensive Agents: Verapamil administered concomitantly with oral antihypertensive agents (e.g., vasodilators, angiotensin-converting enzyme inhibitors, diuretics, beta blockers, prazosin) will usually have an additive effect on lowering blood pressure. Patients receiving these combinations should be appropriately monitored. Disopyramide: Disopyramide should not be administered within 48 hours before or 24 hours after verapamil administration. Quinidine: In patients with hypertrophic cardiomyopathy (IHSS), concomitant use of verapamil and quinidine resulted in significant hypotension. There has been a report of increased quinidine levels during verapamil therapy Nitrates: The pharmacologic profile of verapamil and nitrates as well as clinical experience suggest beneficial interactions. Cimetidine: Two clinical trials have shown a lack of significant verapamil interaction with cimetidine. A third study showed cimetidine reduced verapamil clearance and increased elimination to 1/2. Anesthetic Agents: Verapamil may potentiate the activity of neuromuscular blocking agents and inhalation anesthetics. Carbamazepine: Verapamil may increase carbamazepine concentrations during combined therapy. Rifampin: Therapy with rifampin may markedly reduce oral verapamil bioavailability. Lithium: Verapamil may lower lithium levels in patient on chronic oral lithium therapy. Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no evidence of a carcinogenic potential of verapamil administered to rats for two years. Verapamil was not mutagenic in the Ames test. Studies in female rats did not show impaired fertility. Effects on male fertility have not been determined. Pregnancy (Category C): There are no adequate and well-controlled studies in pregnant women. ISOPTIN crosses the placental barrier and can be detected in umbilical vein blood at delivery. This drug should be used during pregnancy, labor, and delivery, only if clearly needed. Nursing Mothers: ISOPTIN is excreted in human milk, therefore, nursing should be discontinued while verapamil is administered. Pediatric Use: Safety and efficacy of ISOPTIN in children below the age of 18 years have not been established.

ADVERSE REACTIONS: Constipation 8.4%, dizziness 3.5%, nausea 2.7%, hypotension 2.5%, edema 2.1%, headache 1.9%, CHF/pulmonary edema 1.8%, fatigue 1.7%, bradycardia 1.4%, 3° AV block 0.8%, flushing 0.1%, elevated liver enzymes (see WARNINGS). The following reactions, reported in less than 1.0% of patients, occurred under conditions (open trials, marketing experience) where a causal relationship is uncertain; they are mentioned to alert the physician to a possible relationship: angina pectoris, arthralgia and rash, AV block, blurred vision, cerebrovascular accident, chest pain, claudication, confusion, diarrhea, dry mouth, dyspnea, ecchymosis or bruising, equilibrium disorders, exanthema, gastrointestinal distress, gingival hyperplasia, gynecomastia, hair loss, hyperkeratosis, impotence, increased urination, insomnia, macules, muscle cramps, myocardial infarction, palpitations, paresthesia, psychotic symptoms, purpura (vasculitis), shakiness, somnolence, spotty menstruation, sweating, syncope, urticaria. Treatment of Acute Cardiovascular Adverse Reactions: Whenever severe hypotension or complete AV block occur following oral administration of verapamil, the appropriate emergency measures should be applied immediately, e.g., intravenously administered isoproterenol HCl, levarterenol bitartrate, atropine (all in the usual doses), or calcium gluconate (10% solution). If further support is necessary, inotropic agents (dopamine or dobutamine) may be administered. Actual treatment and dosage should depend on the severity and the clinical situation and the judgment and experience of the treating physician.

OVERDOSAGE: Treatment of overdosage should be supportive. Beta-adrenergic stimulation or parenteral administration of calcium solutions may increase calcium ion flux across the slow channel, and have been used effectively in treatment of deliberate overdosage with verapamil Clinically significant hypotensive reactions or fixed high degree AV block should be treated with vasopressor agents or cardiac pacing, respectively Asystole should be handled by the usual measures including cardiopulmonary resuscitation.

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Surgeon General's Report on ACQUIRED IMMUNE DEFICIENCY SYNDROME

A I D S

Foreword

This is a report from the Surgeon General of the U.S. Public Health Service to the people of the United States on AIDS. Acquired Immune Deficiency Syndrome is an epidemic that has already killed thousands of people, mostly young, productive Americans. In addition to illness, disability, and death, AIDS has brought fear to the hearts of most Americans – fear of disease and fear of the unknown. Initial reporting of AIDS occurred in the United States, but AIDS and the spread of the AIDS virus is an international problem. This report focuses on prevention that could be applied in all countries.

My report will inform you about AIDS, how it is transmitted, the relative risks of infection and how to prevent it. It will help you understand your fears. Fear can be useful when it helps people avoid behavior that puts them at risk for AIDS. On the other hand, unreasonable fear can be as crippling as the disease itself. If you are participating in activities that could expose you to the AIDS virus, this report could save your life.

In preparing this report, I consulted with the best medical and scientific experts this country can offer. I met with leaders of organizations concerned with health, education, and other aspects of our society to gain their views of the problems associated with AIDS. The information in this report is current and timely.

This report was written personally by me to provide the necessary understanding of AIDS.

The vast majority of Americans are against illicit drugs. As a health officer I am opposed to the use of illicit drugs. As a practicing physician for more than forty years, I have seen the devastation that follows the use of illicit drugs – addiction, poor health, family disruption, emotional disturbances and death. I applaud the President's initiative to rid this nation of the curse of illicit drug use and addiction. The success of his initiative is critical to the health of the American people and will also help reduce the number of persons exposed to the AIDS virus.

Some Americans have difficulties in dealing with the subjects of sex, sexual practices, and alternate lifestyles. Many Americans are opposed to homosexuality, promiscuity of any kind, and prostitution. This report must deal with all of these issues, but does so with the intent that information and education can change individual behavior, since this is the primary way to stop the epidemic of AIDS. This report deals with the positive and negative consequences of activities and behaviors from a health and medical point of view.

Adolescents and pre-adolescents are those whose behavior we wish to especially influence because of their vulnerability when they are exploring their own sexuality (heterosexual and homosexual) and perhaps experimenting with drugs. Teenagers often consider themselves immortal, and these young people may be putting themselves at great risk.

Education about AIDS should start in early elementary school and at home so that children can grow up knowing the behavior to avoid to protect themselves from exposure to the AIDS virus. The threat of AIDS can provide an opportunity for parents to instill in their children their own moral and ethical standards.

Those of us who are parents, educators and community leaders, indeed all adults, cannot disregard this responsibility to educate our young. The need is critical and the price of neglect is high. The lives of our young people depend on our fulfilling our responsibility.

AIDS is an infectious disease. It is contagious, but it cannot be spread in the same manner as a common cold or measles or chicken pox. It is contagious in the same way that sexually transmitted diseases, such as syphilis and gonorrhea, are contagious. AIDS can also be spread through the sharing of intravenous drug needles and syringes used for injecting illicit drugs.

AIDS is *not* spread by common everyday contact but by sexual contact (penis-vagina, penis-rectum, mouth-rectum, mouth-vagina, mouth-penis). Yet there is great misunderstanding resulting in unfounded fear that AIDS can be spread by casual, non-sexual contact. The first cases of AIDS were reported in this country in 1981. We would know by now if AIDS were passed by casual, non-sexual contact.

Today those practicing high risk behavior who become infected with the AIDS virus are found mainly among homosexual and bisexual men and male and female intravenous drug users. Heterosexual transmission is expected to account for an increasing proportion of those who become infected with the AIDS virus in the future.

At the beginning of the AIDS epidemic many Americans had little sympathy for people with AIDS. The feeling was that somehow people from certain groups "deserved" their illness. Let us put those feelings behind us. We are fighting a disease, not people. Those who are already afflicted are sick people and need our care as do all sick patients. The country must face this epidemic as a unified society. We must prevent the spread of AIDS while at the same time preserving our humanity and intimacy.

AIDS is a life-threatening disease and a major public health issue. Its impact on our society is and will continue to be devastating. By the end of 1991, an estimated 270,000 cases of AIDS will have occurred with 179,000 deaths within the decade since the disease was first recognized. In the year 1991, an estimated 145,000 patients with AIDS will need health and supportive services at a total cost of between $8 and $16 billion. However, AIDS is preventable. It can be controlled by changes in personal behavior. It is the responsibility of every citizen to be informed about AIDS and to exercise the appropriate preventive measures. This report will tell you how.

The spread of AIDS can and must be stopped.

C Everett Koop

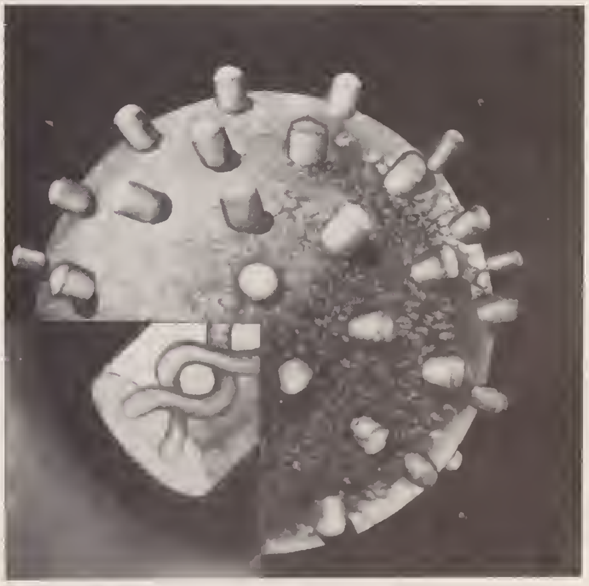
C. Everett Koop, M.D., Sc.D.
Surgeon General

AIDS

AIDS

AIDS Caused by Virus

The letters A-I-D-S stand for Acquired Immune Deficiency Syndrome. When a person is sick with AIDS, he/she is in the final stages of a series of health prob-



Artists drawing of AIDS virus with cut away view showing genetic (reproductive) material.

lems caused by a virus (germ) that can be passed from one person to another chiefly during sexual contact or through the sharing of intravenous drug needles and syringes used for "shooting" drugs. Scientists have named the AIDS virus "HIV or HTLV-III or LAV"[1]. These abbreviations stand for information denoting a virus that attacks white blood cells (T-Lymphocytes) in the human blood. Throughout this publication, we will call the virus the "AIDS virus." The AIDS virus attacks a person's immune system and damages his/her ability to fight other disease. Without a functioning immune system to ward off other germs, he/she now becomes vulnerable to becoming infected by bacteria, protozoa, fungi, and other viruses and malignancies, which may cause life-threatening illness, such as pneumonia, meningitis, and cancer.

No Known Cure

There is presently no cure for AIDS. There is presently no vaccine to prevent AIDS.

Virus Invades Blood Stream

When the AIDS virus enters the blood stream, it begins to attack certain white blood cells (T-Lymphocytes). Substances called antibodies are produced by the body. These antibodies can be detected in the blood by a simple test, usually two weeks to three months after infection. Even before the antibody test is positive, the victim can pass the virus to others by methods that will be explained.

Once an individual is infected, there are several possibilities. Some people may remain well but even so they are able to infect others. Others may develop a disease that is less serious than AIDS referred to as AIDS Related Complex (ARC). In some people the protective immune system may be destroyed by the virus and then other germs (bacteria, protozoa, fungi and other viruses) and cancers that ordinarily would never get a foothold cause "opportunistic diseases" — using the *opportunity* of lowered resistance to infect and destroy. Some of the most common are *Pneumocystis carinii* pneumonia and tuberculosis. Individuals infected with the AIDS virus may also develop certain types of cancers such as Kaposi's sarcoma. These infected people have classic AIDS. Evidence shows that the AIDS virus may also attack the nervous system, causing damage to the brain.

Signs and Symptoms

No Signs

Some people remain apparently well after infection with the AIDS virus. They may have no physically apparent symptoms of illness. However, if proper precautions are not used with sexual contacts and/or intravenous drug use, these infected individuals can spread the virus to others. Anyone who thinks he or she is infected or involved in high risk behaviors should not donate his/her blood, organs, tissues, or sperm because they may now contain the AIDS virus.

ARC

AIDS-Related Complex (ARC) is a condition caused by the AIDS virus in which the patient tests positive for AIDS infection and has a specific set of clinical symptoms. However, ARC patients' symptoms are often less severe than those with the disease we call classic AIDS. Signs and symptoms of ARC may include loss of appetite, weight loss, fever, night sweats, skin rashes, diarrhea, tiredness, lack of resistance to infection, or swollen lymph nodes. These are also signs and symptoms of many other diseases and a physician should be consulted.

AIDS

Only a qualified health professional can diagnose AIDS, which is the result of a natural progress of infection by the AIDS virus. AIDS destroys the body's immune (defense) system and allows otherwise controllable infections to invade the body and cause additional diseases. These opportunistic diseases would not otherwise gain a foothold in the body. These opportunistic diseases may eventually cause death.

Some symptoms and signs of AIDS and the "opportunistic infections" may include a persistent cough and fever associated with shortness of breath or difficult breathing and may be the symptoms of *Pneumocystis carinii* pneumonia. Multiple purplish blotches and bumps on the skin may be a sign of Kaposi's sarcoma. The AIDS virus in all infected people is essentially the same; the reactions of individuals may differ.

Long Term

The AIDS virus may also attack the nervous system and cause delayed damage to the brain. This damage may take years to develop and the symptoms may show up as

---

[1] These are different names given to AIDS virus by the scientific community:

HIV	—	Human Immunodeficiency Virus
HTLV-III	—	Human T-Lymphotropic Virus Type III
LAV	—	Lymphadenopathy Associated Virus

memory loss, indifference, loss of coordination, partial paralysis, or mental disorder. These symptoms may occur alone, or with other symptoms mentioned earlier.

AIDS: the present situation

The number of people estimated to be infected with the AIDS virus in the United States is about 1.5 million. All of these individuals are assumed to be capable of spreading the virus sexually (heterosexually or homosexually) or by sharing needles and syringes or other implements for intravenous drug use. Of these, an estimated 100,000 to 200,000 will come down with AIDS Related Complex (ARC). It is difficult to predict the number who will develop ARC or AIDS because symptoms sometimes take as long as nine years to show up. With our present knowledge, scientists predict that 20 to 30 percent of those infected with the AIDS virus will develop an illness that fits an accepted definition of AIDS within five years. The number of persons known to have AIDS in the United States to date is over 25,000; of these, about half have died of the disease. Since there is no cure, the others are expected to also eventually die from their disease.

The majority of infected antibody positive individuals who carry the AIDS virus show no disease symptoms and may not come down with the disease for many years, if ever.

No Risk from Casual Contact

There is no known risk of non-sexual infection in most of the situations we encounter in our daily lives. We know that family members living with individuals who have the AIDS virus do not become infected except through sexual contact. There is no evidence of transmission (spread) of AIDS virus by everyday contact even though these family members shared food, towels, cups, razors, even toothbrushes, and kissed each other.

Health Workers

We know even more about health care workers exposed to AIDS patients. About 2,500 health workers who were caring for AIDS patients when they were sickest have been carefully studied and tested for infection with the AIDS virus. These doctors, nurses and other health care givers have been exposed to the AIDS patients' blood, stool and other body fluids. Approximately 750 of these health workers reported possible additional exposure by direct contact with a patient's body fluid through spills or being accidentally stuck with a needle. Upon testing these 750, only 3 who had accidentally stuck themselves with a needle had a positive antibody test for exposure to the AIDS virus. Because health workers had much more contact with patients and their body fluids than would be expected from common everyday contact, it is clear that the AIDS virus is not transmitted by casual contact.

Control of Certain Behaviors Can Stop Further Spread of AIDS

Knowing the facts about AIDS can prevent the spread of the disease. Education of those who risk infecting themselves or infecting other people is the only way we can stop the spread of AIDS. People must be responsible about their sexual behavior and must avoid the use of illicit intravenous drugs and needle sharing. We will describe the types of behavior that lead to infection by the AIDS virus and the personal measures that must be taken for effective protection. If we are to stop the AIDS epidemic, we all must understand the disease — its cause, its nature, and its prevention. *Precautions must be taken.* The AIDS virus infects persons who expose themselves to known risk behavior, such as certain types of homosexual and heterosexual activities or sharing intravenous drug equipment.

Risks

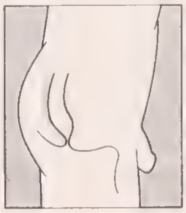
Although the initial discovery was in the homosexual community, AIDS is not a disease only of homosexuals. AIDS is found in heterosexual people as well. AIDS is not a black or white disease. AIDS is not just a male disease. AIDS is found in women; it is found in children. In the future AIDS will probably increase and spread among people who are not homosexual or intravenous drug abusers in the same manner as other sexually transmitted diseases like syphilis and gonorrhea.

Sex Between Men

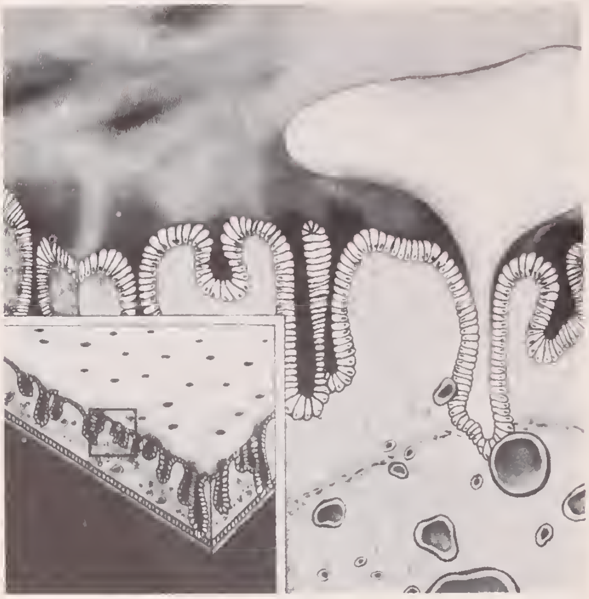
Men who have sexual relations with other men are especially at risk. About 70 percent of AIDS victims throughout the country are male homosexuals and bisexuals. This percentage probably will decline as heterosexual transmission increases. *Infection results from a sexual relationship with an infected person.*

Multiple Partners

The risk of infection increases according to the number of sexual partners one has, *male or female.* The more partners you have, the greater the risk of becoming infected with the AIDS virus.



Vulnerable rectum lining provides avenue for entry of AIDS virus into the blood stream.



How Exposed

Although the AIDS virus is found in several body fluids, a person acquires the virus during sexual contact with an infected person's blood or semen and possibly vaginal secretions. The virus then enters a person's blood stream through their rectum, vagina or penis.

Small (unseen by the naked eye) tears in the surface lining of the vagina or rectum may occur during insertion of the penis, fingers, or other objects, thus opening an avenue for entrance of the virus directly into the blood stream; therefore, the AIDS virus can be passed from penis to rectum and vagina and vice versa without a visible tear in the tissue or the presence of blood.

Prevention of Sexual Transmission – Know Your Partner

Couples who maintain mutually faithful monogamous relationships (only one continuing sexual partner) are protected from AIDS through sexual transmission. If you have been faithful for at least five years and your partner has been faithful too, neither of you is at risk. If you have not been faithful, then you and your partner are at risk. If your partner has not been faithful, then your partner is at risk which also puts you at risk. This is true for both heterosexual and homosexual couples. Unless it is possible to know with *absolute certainty* that neither you nor your sexual partner is carrying the virus of AIDS, you must use protective behavior. *Absolute certainty* means not only that you and your partner have maintained a mutually faithful monogamous sexual relationship, but it means that neither you nor your partner has used illegal intravenous drugs.

AIDS: you can protect yourself from infection

Some personal measures are adequate to safely protect yourself and others from infection by the AIDS virus and its complications. Among these are:

- If you have been involved in any of the high risk sexual activities described above or have injected illicit intravenous drugs into your body, you should have a blood test to see if you have been infected with the AIDS virus.

- If your test is positive or if you engage in high risk activities and choose not to have a test, you should tell your sexual partner. If you jointly decide to have sex, you must protect your partner by always using a rubber (condom) during (start to finish) sexual intercourse (vagina or rectum).

- If your partner has a positive blood test showing that he/she has been infected with the AIDS virus or you suspect that he/she has been exposed by previous heterosexual or homosexual behavior or use of intravenous drugs with shared needles and syringes, a rubber (condom) should always be used during (start to finish) sexual intercourse (vagina or rectum).

- If you or your partner is at high risk, avoid mouth contact with the penis, vagina, or rectum.
- Avoid all sexual activities which could cause cuts or tears in the linings of the rectum, vagina, or penis.
- Single teen-age girls have been warned that pregnancy and contracting sexually transmitted diseases can be the result of only one act of sexual intercourse. They have been taught to say *NO* to sex! They have been taught to say *NO* to drugs! By saying *NO* to sex and drugs, they can avoid AIDS which can *kill* them! The same is true for teenage boys who should also not have rectal intercourse with other males. It may result in AIDS.
- Do not have sex with prostitutes. Infected male and female prostitutes are frequently also intravenous drug abusers; therefore, they may infect clients by sexual intercourse and other intravenous drug abusers by sharing their intravenous drug equipment. Female prostitutes also can infect their unborn babies.

Dirty intravenous needle and syringe contaminated with blood that may contain the AIDS virus.

Intravenous Drug Users

Drug abusers who inject drugs into their veins are another population group at high risk and with high rates of infection by the AIDS virus. Users of intravenous drugs make up 25 percent of the cases of AIDS throughout the country. The AIDS virus is carried in contaminated blood left in the needle, syringe, or other drug related implements and the virus is injected into the new victim by reusing dirty syringes and needles. Even the smallest amount of infected blood left in a used needle or syringe can contain live AIDS virus to be passed on to the next user of those dirty implements.

No one should shoot up drugs because addiction, poor health, family disruption, emotional disturbances and death could follow. However, many drug users are addicted to drugs and for one reason or another have not changed their behavior. For these people, the only way not to get AIDS is *to use a clean, previously unused* needle, syringe or any other implement necessary for the injection of the drug solution.

Hemophilia

Some persons with hemophilia (a blood clotting disorder that makes them subject to bleeding) have been infected with the AIDS virus either through blood transfusion or the use of blood products that help their blood clot. Now that we know how to prepare safe blood products to aid clotting, this is unlikely to happen. This group represents a very small percentage of the cases of AIDS throughout the country.

Blood Transfusion

Currently all blood donors are initially screened and blood

is *not* accepted from high risk individuals. Blood that has been collected for use is tested for the presence of antibody to the AIDS virus. However, some people may have had a blood transfusion prior to March 1985 before we knew how to screen blood for safe transfusion and may have become infected with the AIDS virus. Fortunately there are not now a large number of these cases. With routine testing of blood products, the blood supply for transfusion is now safer than it has ever been with regard to AIDS.

Persons who have engaged in homosexual activities or have shot street drugs within the last 10 years should *never* donate blood.

Mother Can Infect Newborn

If a woman is infected with the AIDS virus and becomes pregnant, she is more likely to develop ARC or classic AIDS, and she can pass the AIDS virus to her unborn child. Approximately one third of the babies born to AIDS-infected mothers will also be infected with the AIDS virus. Most of the infected babies will eventually develop the disease and die. Several of these babies have been born to wives of hemophiliac men infected with the AIDS virus by way of contaminated blood products. Some babies have also been born to women who became infected with the AIDS virus by bisexual partners who had the virus. Almost all babies with AIDS have been born to women who were intravenous drug users or the sexual partners of intravenous drug users who were infected with the AIDS virus. More such babies can be expected.

Think carefully if you plan on becoming pregnant. If there is any chance that you may be in any high risk group or that you have had sex with someone in a high risk group, such as homosexual and bisexual males, drug abusers and their sexual partners, see your doctor.

Summary

AIDS affects certain groups of the population. Homosexual and bisexual males who have had sexual contact with other homosexual or bisexual males as well as those who "shoot" street drugs are at greatest risk of exposure, infection and eventual death. Sexual partners of these high risk individuals are at risk, as well as any children born to women who carry the virus. Heterosexual persons are increasingly at risk.

AIDS: what is safe

Most Behavior is Safe

Everyday living does not present any risk of infection. You *cannot* get AIDS from casual social contact. Casual social contact should not be confused with casual *sexual* contact which is a major cause of the spread of the AIDS virus. Casual *social* contact such as shaking hands, hugging, social kissing, crying, coughing or sneezing, will not transmit the AIDS virus. Nor has AIDS been contracted from swimming in pools or bathing in hot tubs or from eating in restaurants (even if a restaurant worker has AIDS or carries the AIDS virus.) AIDS is not contracted from sharing bed linens, towels, cups, straws, dishes, or any other eating utensils. You cannot get AIDS from toilets, doorknobs, telephones, office machinery, or household furniture. You cannot get AIDS from body massages, masturbation or any non sexual contact.

Donating Blood

Donating blood is *not* risky at all. *You cannot get AIDS by donating blood.*

Receiving Blood

In the U.S. every blood donor is screened to exclude high risk persons and every blood donation is now tested for the presence of antibodies to the AIDS virus. Blood that shows exposure to the AIDS virus by the presence of antibodies is not used either for transfusion or for the manufacture of blood products. Blood banks are as safe as current technology can make them. Because antibodies do not form immediately after exposure to the virus, a newly infected person may unknowingly donate blood after becoming infected but before his/her antibody test becomes positive. It is estimated that this might occur less than once in 100,000 donations.

There is no danger of AIDS virus infection from visiting a doctor, dentist, hospital, hairdresser or beautician. AIDS cannot be transmitted non-sexually from an infected person through a health or service provider to another person. Ordinary methods of disinfection for urine, stool and vomitus which are used for non-infected people are adequate for people who have AIDS or are carrying the AIDS virus. You may have wondered why your dentist wears gloves and perhaps a mask when treating you. This does not mean that he has AIDS or that he thinks you do. He is protecting you and himself from hepatitis, common colds or flu.

There is no danger in visiting a patient with AIDS or caring for him or her. Normal hygienic practices, like wiping of body fluid spills with a solution of water and household bleach (1 part household bleach to 10 parts water), will provide full protection.

Children in School

None of the identified cases of AIDS in the United States are known or are suspected to have been transmitted from one child to another in school, day care, or foster care settings. Transmission would necessitate exposure of open cuts to the blood or other body fluids of the infected child, a highly unlikely occurrence. Even then routine safety procedures for handling blood or other body fluids (which should be standard for all children in the school or day care setting) would be effective in preventing transmission from children with AIDS to other children in school.

Children with AIDS are highly susceptible to infections, such as chicken pox, from other children. Each child with AIDS should be examined by a doctor before attending school or before returning to school, day care or foster care settings after an illness. No blanket rules can be made for all school boards to cover all possible cases of children with AIDS and each case should be considered separately and individualized to the child and the setting, as would be done with any child with a special problem, such as cerebral palsy or asthma. A good team to make such decisions with the school board would be the child's parents, physician and a public health official.

Casual social contact between children and persons infected with the AIDS virus is not dangerous.

Insects

There are no known cases of AIDS transmission by insects, such as mosquitoes.

Pets

Dogs, cats and domestic animals are not a source of infection from AIDS virus.

Tears and Saliva

Although the AIDS virus has been found in tears and saliva, no instance of transmission from these body fluids has been reported.

AIDS comes from sexual contacts with infected persons and from the sharing of syringes and needles. There is no danger of infection with AIDS virus by casual social contact.

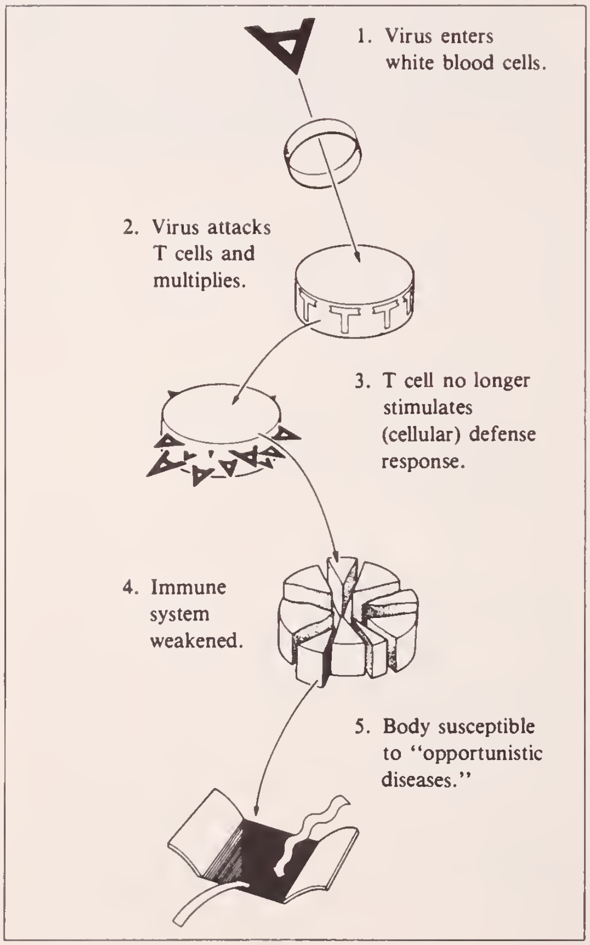
Testing of Military Personnel

You may wonder why the Department of Defense is currently testing its uniformed services personnel for presence of the AIDS virus antibody. The military feel this procedure is necessary because the uniformed services act as their own blood bank in a time of national emergency. They also need to protect new recruits (who unknowingly may be AIDS virus carriers) from receiving live virus vaccines. These vaccines could activate disease and be potentially life-threatening to the recruits.

AIDS: what is currently understood

Although AIDS is still a mysterious disease in many ways, our scientists have learned a great deal about it. In five years we know more about AIDS than many diseases that we have studied for even longer periods. While there is no vaccine or cure, the results from the health and behavioral research community can only add to our knowledge and increase our understanding of the disease and ways to prevent and treat it.

In spite of all that is known about transmission of the AIDS virus, scientists will learn more. One possibility is the potential discovery of factors that may better explain the mechanism of AIDS infection.


1. Virus enters white blood cells.
2. Virus attacks T cells and multiplies.
T T T
3. T cell no longer stimulates (cellular) defense response.
4. Immune system weakened.
5. Body susceptible to "opportunistic diseases."

Why are the antibodies produced by the body to fight the AIDS virus not able to destroy that virus?

The antibodies detected in the blood of carriers of the AIDS virus are ineffective, at least when classic AIDS is actually triggered. They cannot check the damage caused by the virus, which is by then present in large numbers in the body. Researchers cannot explain this important observation. We still do not know why the AIDS virus is not destroyed by man's immune system.

Summary

AIDS no longer is the concern of any one segment of society; it is the concern of us all. No American's life is in danger if he/she or their sexual partners do not engage in high risk sexual behavior or use shared needles or syringes to inject illicit drugs into the body.

People who engage in high risk sexual behavior or who shoot drugs are risking infection with the AIDS virus and are risking their lives and the lives of others, including their unborn children.

We cannot yet know the full impact of AIDS on our society. From a clinical point of view, there may be new manifestations of AIDS — for example, mental disturbances due to the infection of the brain by the AIDS virus in carriers of the virus. From a social point of view, it may bring to an end the free-wheeling sexual lifestyle which has been called the sexual revolution. Economically, the care of AIDS patients will put a tremendous strain on our already overburdened and costly health care delivery system.

The most certain way to avoid getting the AIDS virus and to control the AIDS epidemic in the United States is for individuals to avoid promiscuous sexual practices, to maintain mutually faithful monogamous sexual relationships and to avoid injecting illicit drugs.

Look to the Future

The Challenge of the Future

An enormous challenge to public health lies ahead of us and we would do well to take a look at the future. We must be prepared to manage those things we can predict, as well as those we cannot.

At the present time there is no vaccine to prevent AIDS. There is no cure. AIDS, which can be transmitted sexually and by sharing needles and syringes among illicit intravenous drug users, is bound to produce profound changes in our society, changes that will affect us all.

Information and Education Only Weapons Against AIDS

It is estimated that in 1991 54,000 people will die from AIDS.

At this moment, many of them are not infected with the AIDS virus. With proper information and education, as many as 12,000 to 14,000 people could be saved in 1991 from death by AIDS.

AIDS will Impact All

The changes in our society will be economic and political and will affect our social institutions, our educational practices, and our health care. Although AIDS may never touch you personally,the societal impact certainly will.

Be Educated – Be Prepared

Be prepared. Learn as much about AIDS as you can. Learn to separate scientific information from rumor and myth. The Public Health Service, your local public health officials and your family physician will be able to help you.

Concern About Spread of AIDS

While the concentration of AIDS cases is in the larger urban areas today, it has been found in every state and with the mobility of our society, it is likely that cases of AIDS will appear far and wide.

Special Educational Concerns

There are a number of people, primarily adolescents, that do not yet know they will be homosexual or become drug abusers and will not heed this message; there are others who are illiterate and cannot heed this message. They must be reached and taught the risk behaviors that expose them to infection with the AIDS virus.

High Risk Get Blood Test

The greatest public health problem lies in the large number of individuals with a history of high risk behavior who have been infected with and may be spreading the AIDS virus. Those with high risk behavior must be encouraged to protect others by adopting safe sexual practices and by the use of clean equipment for intravenous drug use. If a blood test for antibodies to the AIDS virus is necessary to get these individuals to use safe sexual practices, they should get a blood test. Call your local health department for information on where to get the test.

Anger and Guilt

Some people afflicted with AIDS will feel a sense of anger and others a sense of guilt. In spite of these understandable reactions, everyone must join the effort to control the epidemic, to provide for the care of those with AIDS, and to do all we can to inform and educate others about AIDS, and how to prevent it.

Confidentiality

Because of the stigma that has been associated with AIDS, many afflicted with the disease or who are infected with the AIDS virus are reluctant to be identified with AIDS. Because there is no vaccine to prevent AIDS and no cure, many feel there is nothing to be gained by revealing sexual contacts that might also be infected with the AIDS virus. When a community or a state requires reporting of those infected with the AIDS virus to public health authorities in order to trace sexual and intravenous drug contacts—as is the practice with other sexually transmitted diseases—those infected with the AIDS virus go underground out of the mainstream of health care and education. For this reason current public health practice is to protect the privacy of the individual infected with the AIDS virus and to maintain the strictest confidentiality concerning his/her health records.

State and Local AIDS Task Forces

Many state and local jurisdictions where AIDS has been seen in the greatest numbers have AIDS task forces with heavy representation from the field of public health joined by others who can speak broadly to issues of access to care, provision of care and the availability of community and psychiatric support services. Such a task force is needed in every community with the power to develop plans and policies, to speak, and to act for the good of the public health at every level.

State and local task forces should plan ahead and work collaboratively with other jurisdictions to reduce transmission of AIDS by far-reaching informational and educational programs. As AIDS impacts more strongly on society, they should be charged with making recommendations to provide for the needs of those afflicted with AIDS. They also will be in the best position to answer the concerns and direct the activities of those who are not infected with the AIDS virus.

The responsibility of State and local task forces should be far reaching and might include the following areas:

- Insure enforcement of public health regulation of such

practices as ear piercing and tattooing to prevent transmission of the AIDS virus.
- Conduct AIDS education programs for police, firemen, correctional institution workers and emergency medical personnel for dealing with AIDS victims and the public.
- Insure that institutions catering to children or adults who soil themselves or their surroundings with urine, stool, and vomitus have adequate equipment for cleanup and disposal, and have policies to insure the practice of good hygiene.

School

Schools will have special problems in the future. In addition to the guidelines already mentioned in this pamphlet, there are other things that should be considered such as sex education and education of the handicapped.

Sex Education

Education concerning AIDS must start at the lowest grade possible as part of any health and hygiene program. The appearance of AIDS could bring together diverse groups of parents and educators with opposing views on inclusion of sex education in the curricula. There is now no doubt that we need sex education in schools and that it must include information on heterosexual and homosexual relationships. The threat of AIDS should be sufficient to permit a sex education curriculum with a heavy emphasis on prevention of AIDS and other sexually transmitted diseases.

Handicapped and Special Education

Children with AIDS or ARC will be attending school along with others who carry the AIDS virus. Some children will develop brain disease which will produce changes in mental behavior. Because of the right to special education of the handicapped and the mentally retarded, school boards and higher authorities will have to provide guidelines for the management of such children on a case-by-case basis.

Labor and Management

Labor and management can do much to prepare for AIDS so that misinformation is kept to a minimum. Unions should issue preventive health messages because many employees will listen more carefully to a union message than they will to one from public health authorities.

AIDS Education at the Work Site

Offices, factories, and other work sites should have a plan in operation for education of the work force and accommodation of AIDS or ARC patients *before* the first such case appears at the work site. Employees with AIDS or ARC should be dealt with as are any workers with a chronic illness. In-house video programs provide an excellent source of education and can be individualized to the needs of a specific work group.

Strain on the Health Care Delivery System

The health care system in many places will be overburdened as it is now in urban areas with large numbers of AIDS patients. It is predicted that during 1991 there will be 145,000 patients requiring hospitalization at least once and 54,000 patients who will die of AIDS. Mental disease (dementia) will occur in some patients who have the AIDS virus before they have any other manifestation such as ARC or classic AIDS.

State and local task forces will have to plan for these patients by utilizing conventional and time honored systems but will also have to investigate alternate methods of treatment and alternate sites for care including homecare.

The strain on the health system can be lessened by family, social, and psychological support mechanisms in the community. Programs are needed to train chaplains, clergy, social workers, and volunteers to deal with AIDS. Such support is particularly critical to the minority communities.

Mental Health

Our society will also face an additional burden as we better understand the mental health implications of infection by the AIDS virus. Upon being informed of infection with the AIDS virus, a young, active, vigorous person faces anxiety and depression brought on by fears associated with social isolation, illness, and dying. Dealing with these individual and family concerns will require the best efforts of mental health professionals.

Controversial Issues

A number of controversial AIDS issues have arisen and will continue to be debated largely because of lack of knowledge about AIDS, how it is spread, and how it can be prevented. Among these are the issues of compulsory blood testing, quarantine, and identification of AIDS carriers by some visible sign.

Compulsory Blood Testing

Compulsory blood testing of individuals is not necessary. The procedure could be unmanageable and cost prohibitive. It can be expected that many who *test* negatively might actually be positive due to *recent* exposure to the AIDS virus and give a false sense of security to the individual and his/her sexual partners concerning necessary protective behavior. The prevention behavior described in this report, if adopted, will protect the American public and contain the AIDS epidemic. Voluntary testing will be available to those who have been involved in high risk behavior.

Quarantine

Quarantine has no role in the management of AIDS because AIDS is not spread by casual contact. The only time that some form of quarantine might be indicated is in a situation where an individual carrying the AIDS virus knowingly and willingly continues to expose others through sexual contact or sharing drug equipment. Such circumstances should be managed on a case-by-case basis by local authorities.

Identification of AIDS Carriers by Some Visible Sign

Those who suggest the marking of carriers of the AIDS virus by some visible sign have not thought the matter through thoroughly. It would require testing of the entire population which is unnecessary, unmanageable and costly. It would miss those recently infected individuals who would test negatively, but be infected. The entire procedure would give a false sense of security. AIDS must and will be treated as a disease that can infect anyone. AIDS should not be used as an excuse to discriminate against any group or individual.

Updating Information

As the Surgeon General, I will continually monitor the most current and accurate health, medical, and scientific information and make it available to you, the American people. Armed with this information you can join in the discussion and resolution of AIDS-related issues that are critical to your health, your children's health, and the health of the nation.

History of Medicine in Alaska

VERNON A. CATES, M.D.

Vernon Cates was born on a national holiday, February 12, 1920, in Seattle. He spent much of his childhood, however, in the tri-city area of eastern Washington, returning to his birthplace to graduate from Franklin High School.

He entered the University of Washington. Having had little science in high school, he arrived at the University seeking the broadest field he could find. His five closest friends were going into law. He liked art but decided that would be a better avocation. He flipped a coin to decide whether to follow friends or try premed.

The flip of the coin led him to the then probably 75-year old Dr. Wooster's class attended by close to five hundred students. Dr. Wooster declared that out of this group 10 would become physicians. Vern wondered what he had done. He picked his own courses, anatomy and quantitative analysis among them. In his third year, World War II started and many of the instructors were drafted. Consequently, Dr. Wooster picked him to be first assistant in anatomy. It was as if, "Here's a guy with one-half a body's experience." Vern managed to teach but felt that he merely kept a few pages ahead of his students.

He finished the University taking courses and teaching. After graduation, he stayed an extra year to teach. Vern found himself extremely busy since the regular students were on semesters, and the V-12 students[1] were on a quarter system. He was teaching histology, embryology, neurology, and anatomy. "Spare time" was taken up with being a premedical advisor.

One of his students was Bill Maddock who was to graduate from medical school at a very young age and who was to begin research in his first year of medical school. Vern advised Bill to take something other than what Vern was teaching.

As well as premed students, he taught nursing students; one of whom was Grace Moore. He does not pretend to remember teaching her or perhaps he pretends not to remember. She had grown up in Chehalis, a lovely farming valley south of Seattle. Graduating from Napavine High School, she entered the University of Washington to receive a B.S. in nursing.

Eventually, Vern was to go to medical school with at least forty others whom he had taught. He entered the University of Oregon Medical School[2] because at this time the University of Washington had no medical school. In fact, Dr. Wooster, his mentor, had been brought to Washington to start basic sciences with the goal of establishing a medical school—a goal that was only realized in 1951.

He interned at King County Hospital where he was sent to Grace's floor—she was head nurse in medicine. Following his internship, Vern had planned to travel to France with a musician friend to study art. He married Gracie instead. He had visited Juneau but was not comfortable with the practice there. However, he met Dr. Ray Coffin who was starting the Doctors Clinic in Anchorage. Dr. Coffin was the reason Vern decided to come to Anchorage, sight unseen. Grace and he were married on August 27, 1948, the day before the Alaska steamship sailed.

In addition to the new Doctors Clinic consisting of Drs. Sogn, Coffin, Brandon and Russ Jackson, there were seven other doctors in Anchorage: Drs. Romig, Martin, George Davis, Fritz, O'Malley, Virginia Wright[3] and Walkowski. Dr. Flora had been killed that year in a plane crash.

Vern went to Palmer a year after his arrival to establish a branch of the clinic there and to encourage a physician who was practicing extremely questionable medicine to leave the territory of Alaska. He left. They stayed for the year, which Grace remembers fondly, because everyone was so friendly. If Gracie had been unhappy to go to the valley, she was equally unhappy to leave it.

In the meantime, Bill Ivy, who had interned with Vern, had started a branch clinic in Spenard. To round things out, Dr. Merritt Starr, who had been a resident during Vern's internship, joined the clinic.

When the Cates' returned, they went into the beginnings of their house on Wildwood Lane, overlooking Chester Flats and, on the horizon, the city. It was then considered *far out* of town. The house consisted of two rooms, a bath and a closet-sized room. The basement was rented to a military couple, because housing was difficult to find. The house was scarcely large enough for two people, and there were two Cates' children by now. The house, started in 1939, had acquired the first telephone in Spenard. The telephone became so popular that eventually thirty people were on the line. In over twenty-five years, the Cates built onto the house 11 times.

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[1] A Navy program to allow medical students to earn a degree, followed by two years minimum in the service.

[2] Now the Oregon Health Sciences School.

[3] Mrs. James O'Malley

Vern claims that Dr. Sogn made a surgeon of him. Vern praises his skill and speed. The first week, Dr. Sogn asked him to assist at a gall bladder operation. At the next gall bladder operation, Dr. Sogn declared: "I'm going to be the assistant." After 3 minutes, he stated, "You assist. I'll be scrub nurse." After 5 minutes, "I think I'll go have some coffee."

One day Dr. Sogn asked, "Have you ever taken out a kidney?" When Vern nodded, "No," Sogn said, "Neither have I!" Vern studied hard all night. But of course, Dr. Sogn had and did.

Both Dr. Coffin and Dr. Sogn were good at training young physicians. Sogn did the surgery, Coffin kept them alive, and Vern had the experience.

Vern Cates had much respect for Howard Romig and other early physicians in the area. One night, he found Howard immobilized on a sofa at the hospital[4]. Howard asked him to go see a patient who had an acute abdomen. Howard wanted his opinion. When Vern said he thought it was an appendix, Howard replied he thought it was a ruptured ulcer. An hour later, Howard, with the assistance of Asa Martin, had opened up the right lower quadrant but it was not the appendix, so now he was repairing the ulcer.

When the admired and brilliant Bill Maddock died in 1969, it was a loss which Vern Cates still feels. "Maddock made things work," says Vern.

Vern Cates is a sensitive man who keenly feels some of the rough events of medicine in Alaska—Dr. Andrew Ivy and Krebiozen; the building of the new clinic and hospital building at 825 L Street[5]; the malpractice problems of 1976, especially the mandatory and exclusive insurance of MICA which the Doctors Clinic fought; and the eventual breakup of the Alaska Clinic. Vern feels that with twenty-five physicians, the clinic peaked. Things were definitely downhill from then on as its membership grew to sixty plus.

One night he was traveling along L Street, on heavy ice, en route to the Presbyterian Hospital to deliver a baby. As he drove by the Inlet Towers, the "Little Red Wagon" pulled out at great speed. His options were to run head on into the oncoming traffic[7], broadside the ambulance, or drive up the back of the car in front. He chose the latter. He destroyed the trunk of the car, but out of the wreck filed 6 teenagers, all rubbing the backs of their necks. He announced that he would see them at the police station after going to the hospital and went on. His patient had delivered but he was called to the emergency room to pronounce the person in the ambulance dead. Five of the kids voluntarily came to see him, but there was never a hint of a lawsuit.

This incident made him recall that for three years in the 1960's, the insurance cost for the clinic for 20–25 physicians was $3,500. Perhaps this is the most improbable part of the story of the physician who has practiced longer than any doctor in Anchorage. Statewide, he bows to Dr. Rude of Juneau who graduated from medical school in 1928, and on last report, was still seeing a few old patients.

Gwynneth Gminder Wilson

AAPA-OM

AAPA-OM is the professional association for Physicians Assistants who provide health care for workers with job related illnesses and injuries. It is the focus of this organization to promote to its members the highest professional medical standard as they provide medical care to the Nation's workforce.

We promote programs that lead toward a safer and healthier work-place environment through health promotion, management consulting and medical monitoring.

We strive to develop and expand the Physicians Assistants' integral role on the Occupational Health Team through close interface with management, supervising physicians, occupational health nurses, industrial hygienists and safety engineers.

The AAPA-OM (American Academy of Physicians Assistants in Occupational Medicine) seeks members and the exchange of ideas and opportunities. For further information or membership write AAPA-OM, P. O. Box 53164, Oklahoma City, OK 73125.

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[4] Providence Hospital was then located on L Street between 8th and 9th Avenues.

[5] Started as the Presbyterian Hospital, becoming Community Hospital, moved by the Teamsters to a building on DeBarr, now Humana Hospital.

[6] This had become the name of the Doctors Clinic.

[7] L Street was two ways.

President's Page

Our second annual spring lobbying session in Juneau in conjunction with our spring ASMA Council Meeting was at least a modest success. I believe we will succeed in passing legislation to improve funding of the State Medical Board by tying its funding reasonably closely to our license fees which, of course, will need to increase to support appropriate Board functioning.

The progress of Tort Reform Legislation suffers from a few more dips and heaves in the road. We are hoping to secure passage of reasonably strong Tort Reform Legislation in the Alaska State Senate this year, but whether we will see any success in the House during 1987 is doubtful. An encouraging sign, if one may call it that, is that the effect of Alaska Tort Law is being felt in the hinterlands and may be commanding the attention of consumers of health care. There are already a good half-dozen small communities in Alaska where the doctors are unable to afford any liability insurance, which means their patients are without any substantial protection in the event of a real incident of malpractice with significant damages. There are also a number of small communities where obstetrical care either has become unavailable or is restricted, with "high risk" patients being referred out mainly for liability reasons.

Twelve years ago in California, it was an outpouring of letters from patients rather than doctors that pushed the Legislature off dead-center and obtained rapid passage of the Medical Injury Compensation Reform Act which had been stalled in committee. (Does this sound familiar?) Next year may be the year for such a campaign in Alaska. Do not be surprised if your State Medical Association encourages you to talk to your patients about this subject and to urge those patients affected by it to let their legislators know what the tort system is doing to their access to care.

It has been my pleasure to serve you this year! I know that we are looking forward to another successful year with Jerry Little's leadership.

Donald E. Thieman, M.D.
President
Alaska State Medical Association

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Executive Privilege

Juneau or bust! I'm sure that was one of the catch-phrases prospective miners coined as they embarked for Juneau and its offerings of gold. Some found their dream, but many found only fool's gold. Some things never change!

As we completed our days in Juneau lobbying for our interests, it became apparent to me that this sojourn is critically important to the education of our membership. Discussing issues was certainly important, but understanding the process is essential if we are ever to have any impact on the system.

Presently, the medical profession has only one individual on a commission and none on a board of state government. How can change occur unless we participate? How can we respond if we aren't listening? How can we act if we are not aware? We must have the ears with which to listen. Therefore, we encourage each of you to take an active role in your community's political life so that we can begin to develop the listening devices we need to create policy. It is critical that physicians volunteer or suggest colleagues that could serve on state boards or commissions. This will begin the process of involvement—involvement that will give us a voice in the creation of policy rather than our present "voice in the wilderness."

ASMA is developing a five-year plan of action to present to the Council. This plan will effectively "get us in the process" of electing officials to government and of understanding the bureaucracy of state government. Even though your individual political views are as diverse as your specialties, concern about quality of care and the delivery of that care is the binding thread that tightens our interests together.

If any be so foolish as to believe that government will not affect your ability to deliver quality care, then fool's gold is your reward. We are losing physicians in private practice because they cannot afford to deliver quality care. Try as we may, however, we have not been able to alter the course of legislation that would effectively change this state of affairs.

The time is now! I will be calling many of you to assist our efforts over the next five years in the development of a Governmental Affairs Committee. In conjunction with the Council and the Executive Committee, we will plan for the future NOW. We will ask physicians in rural communities to seek qualified candidates within their communities. We will ask that you discuss the issues with your circle of friends and patients. As a respected member of your community, your voice creates attention and response. These are tools we sorely need. In urban areas we will ask physicians to actively attend and support ALPAC functions. We will ask physicians to lend their financial and physical support by attending political gatherings. But, we will never ask you to give up your right to disagree.

So, please join with us *now* by participating in activities that will increase your awareness of government and its effect on your lives. Individually it's difficult to make a difference, but cooperatively we can leap tall buildings—even the Capitol. Our choice for the future seems quite clear, as prospective Legislative miners, we must create our own golden opportunities for the future. Leave the fool's gold for others!

Washington Trip Report

David E. Johnson, M.D.[1]

Doug Smith, M.D., AMA Delegate from Anchorage; Ray Schalow, ASMA Executive Director; and I went to Washington, D.C., for the spring joint lobbying effort with the California and Idaho Medical Associations. We met with Senators Stevens and Murkowski and Representative Young, and with a number of agency personnel. We joined the California group to visit several California Congressmen as well.

Physician Diagnostic Related Groups (DRG's) were on the top of nearly every agenda with folks in Washington. Widely viewed as a trial balloon from the Administration's Office of Management and Budget (OMB), the so-called RAP physician DRG has not even been released in draft legislation form. The AMA is adamantly opposed to this proposal, which would include radiologists, anesthesiologists and pathologists (RAP) in hospital DRG's. The full Alaskan delegation has signed a joint resolution condemning the proposal. I would be very surprised if physician DRG's were enacted this year. I would also be surprised if they were not brought up again next year.

Most people that we talked to believe that there will be some changes in physician reimbursement, but nobody is prepared to claim victory for one specific proposal. Capitation, or a fixed payment per beneficiary through an intermediary, is high on the Administration's agenda, but many other people have a lot of questions about it. The AMA-Harvard Relative Value Scales study report is due in 1988, and many in Congress seem willing to tinker just a little bit and wait to see what that study recommends. There seems to me to be more awareness than before that quality of service concerns must be addressed before any massive changes are made in physician reimbursement.

Secretary Bowen's proposal regarding catastrophic coverage through Medicare has thoroughly impressed Congress, and some form of catastrophic insurance will almost certainly pass the 100th Congress, probably this year. There are several other proposals floating around, most notably the Stark-Gradison proposal from the Chairman and ranking minority member, respectively, of the Health Subcommittee of the House Ways and Means committee. The disagreements now are about what specifically will be covered and how it will be financed. The AMA has taken a position in support of fiscally responsible coverage of catastrophic illness in Medicare.

We spent much of our time with Senator Stevens discussing the dilemmas of funding both custodial and medical care for the elderly, since he is currently addressing that complex of issues with a fifth member of his family. This issue is addressed in an AMA proposal for long-range funding for Medicare, currently known as Report MM of the Board of Trustees. You will hear more about that as time goes on.

Tobacco legislation is gathering momentum in Congress, though few expect that the AMA-endorsed ban on tobacco advertising will pass this year. A second bill would ban all smoking on all domestic commercial aircraft. Another bill would make tobacco advertising and promotion nondeductible as a business expense for tax purposes. What is most likely to happen this session of Congress is an increase in the tobacco Federal excise tax from its current level of 16 cents per pack to something short of double. Significantly, President Reagan is said to be willing to increase this particular tax, possibly as a means of financing catastrophic care under Medicare.

Acquired Immune Deficiency Syndrome (AIDS) awareness in Congress and the Administration is increasing, but many in Congress are woefully ignorant of this looming disaster. The AMA is working hard at informing key members of Congress in particular, and the whole Congress as soon as possible, about what is involved and what is medically appropriate. Look for a real increase in funding addressing AIDS.

Professional Review Organizations (PRO's) are being pushed to be very hard-nosed. At the same time, the master agency within the Department of Health and Human Services (HHS) within which the PRO program resides, the Health Care Financing Administration (HCFA), reported that they are working at making the communications with patients regarding denials more responsible, and making the censure of physicians more appropriate and confined to due process. Anyone who has had problems with APRO should let the ASMA office know, providing as much specific information as possible. Copies of all correspondence and summaries of phone calls exchanged would be most helpful. ASMA will forward them to the appropriate administrative office in HCFA.

The Maximum Allowable Actual Charge (MAAC) program is widely acknowledged as a nightmare of obfuscation and confusion for physicians. Again, specific examples of fiscal intermediary noncooperation or unhelpfulness—in our case in Alaska that means

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[1] Alternate Delegate to the AMA.

Aetna—or needless complexity will be very helpful in dealing with the problem. Please send as much documentation as you can to ASMA for forwarding to the proper officials: simply a photocopy of the entire packet of information you received from Aetna when you requested information on which to base your MAAC would be very useful.

In Senator Murkowski's office we received updated information regarding the Veterans Administration and the Indian Health Service in Alaska. The VA is going ahead with an Anchorage Clinic on Eighth Avenue and will be moving the pharmacy there from Elmendorf AFB. Their current plan is to take over the old Sohio building in Anchorage and bring all VA programs in Anchorage into that one location, including the alcohol and drug abuse programs, the outpatient clinic, the pharmacy, and all administrative functions. That is tentatively planned for 1989.

We also learned about the Alaska Native Health Service (ANHS) Hospital being planned for a site near Providence Hospital and discussed it later at some length with Representative Young. Anchorage Mayor Tony Knowles issued a press release the day before our visit with the senator that announced a three-way land swap among the Municipality of Anchorage, ANHS, and the Tudor Fund Corporation that would enable construction of a 160-bed hospital, a communicable disease research laboratory, an outpatient clinic, and a short-term housing facility. The proposed complex would employ 700—200 more than the current ANHS hospital. We again requested the information we had asked IHS for last year regarding the feasibility of a new ANHS hospital as opposed to creative use of existing Anchorage and other Alaskan hospital capacity, urging our strong commitment to improving access to appropriate health care for all Alaskans.

Some overall impressions and quotable quotes will conclude this report. Dexann Clohen, AMA lobbyist, told us that while working on the trillion dollar budget any amount under one hundred million dollars is regarded as "budget dust." Many folks in Congress have an aversion to tying Medicare to ability to pay or means testing, but Karen Nelson, a staffer for Henry Waxman of California, referred to a proposal for a sliding scale of Medicare fees for catastrophic coverage as "progressive financing." Rather like a "graduated" income tax, wouldn't you say?! In Congress there seems to be some support for a tax increase to pay for social programs, but the Administration is strongly opposed and has widely promised to veto tax increase legislation. An overall assessment regarding Federal health programs would best be stated that things won't be the same as they were before, but they aren't that bad, either.

This touches some of the highlights of four busy days in Washington. We also received information regarding liability insurance, tort reform, Medicaid, uncompensated care and ERISA, child health issues, immunization legislation and other issues. I would be glad to answer any specific questions that anyone has, either by mail or by phone.

Auxiliary News:

Alaska State Medical Association Auxiliary

In this closing update from your State Medical Association Auxiliary, I'd like to jog your memory for a moment. Recall how well served you are by your auxiliary and how much positive PR has been achieved benefiting all of us.

More than 50 members we contacted participated in Childrens' Healthfest '87. Those of you who worked the display booths with us realize that our Healthfest was a monumental hit with the public! The team spirit encouraged everyone to give their best effort. Anchorage Medical Society Auxiliary president Alice Samuelson and her team of coordinators deserve a round of applause for turning this adventurous idea into reality!

The State Auxiliary project, assisting the American Lung Association of Alaska with a raffle to keep their Asthma "Champ Camp" afloat, was a success as well. They needed a minimum of $6,000. We raised $7,200. The extra funds will go toward "camperships" to assist financially strapped parents. A tremendous amount of work went into locating the 60 donated gifts, calling members to sell tickets, collecting the ticket stubs, and distributing the prizes . Your auxiliary was awarded the double bar cross citation for the greatest donation in 1986–87 to the Alaska Branch of the American Lung Association.

United Way packets also made their way to your offices due to efforts by our members. Also, many holiday gifts were collected from members attending the annual holiday tea in December. These packages were given in the spirit of the season to men and women staying at Providence House. Hats off to Alice Samuelson for her organizational skills put to work on these two projects!

The AMA–ERF contribution this year came to $3,081.23, a $1,601.23 increase from the year before. A pat on the back to Carolyn Crouch for all she has contributed to reach this point.

Our Legislation Chairman, Cassie Hill, organized the Phone Bank System to notify you and your spouses of three alerts: 1) the issue of funding the Health Science Library, 2) funding for the AIDS Program in the legislature, and 3) tort reform legislation. She also provided for educational activities on tort reform for the auxiliary general membership.

Ray Schalow and Gayle Mathiesen from the Alaska State Medical Association office, along with Dr. Jerry Little and myself, have met monthly this year to insure you will enjoy our convention in Juneau. It has been a pleasure working with them. The ASMA meeting, June 3–7, will speak for itself as you attend all the activities.

I have been proud of this auxiliary more times than I could count in my year as it's president. It has been my privilege to work with these spouses and with you to meet our joint goals.

Patrice Gerster
President, ASMA Auxiliary

Anchorage Medical Society Auxiliary

A very successful and well-attended "Children's Healthfest '87" was held on April 11, 1987 at the Northway Mall in Anchorage. Months of planning by Healthfest chairperson, Jackie Middaugh and her committee leaders Cherie McGuire, Terrie McEvoy, Jane Erkman, Shirley Kennedy, Julie Sprott, and Alice Samuelson, paid off in a fun and educational day. Anchorage children were entertained and educated by a mock-up operating room complete with a surgeon, nutritional snacks cut into cookie shapes from fruit gelatin, dental exams, ENT exams, physical fitness tests, water safety, an ambulance, heart monitoring equipment, sonograms, and a large variety of other displays. There was a continuous line of children to have arm casts applied. Screenings included vision, blood pressure, and height and weight.

The auxiliary booth at the fair distributed handouts dealing with various health problems, the "Ashes to Ashes" anti-smoking poster, and we introduced a new health project — the "Medi-File". The Medi-File is a wallet size card that gives patients a list of all their current medications and allergies. This card can be shown to the patient's physicians or other health care providers to provide them with an up-to-date listing of medications.

Many thanks to our large number of volunteers who made the health fair possible and such a resounding success! Special thanks to the Anchorage Medical Society for their help, Northway Mall for their generosity, and Alaska Health Fair for their expertise.

Alice Samuelson
President, AMS Auxiliary

Editorial

In this issue of *Alaska Medicine,* we have reprinted the landmark Report of the U.S. Surgeon General on Acquired Immune Deficiency Syndrome. Every physician needs to become well educated about AIDS. The Surgeon General's Report is a good place to start. Physicians must be prepared to help lead the fight against AIDS. We must be informed so we can be resources to our patients and our communities.

AIDS — WHAT HAS CHANGED?

- The number of AIDS cases continues to rise. It is now estimated that in 1991, 270,000 Americans will develop AIDS and 179,000 will have died of AIDS. More than 1.5 million Americans are believed now to be infected with the AIDS virus — one half may develop AIDS. Epidemiologic studies have shown that heterosexuals increasingly are becoming infected and that heterosexual transmission of the virus occurs more easily than initially suspected.
- Experts believe it unlikely that a vaccine against AIDS will be available or drugs to cure AIDS will be discovered in the next five years—or even longer.
- The National Research Council and Institute of Medicine of the National Academy of Sciences, after reviewing all available information about AIDS, published a major work—"Confronting AIDS, Directions for Public Health, Health Care, and Research." These experts have called for a major nationwide response to AIDS, recommending federal government expenditures of one billion dollars per year for research and one billion dollars per year for disease prevention education.
- Surgeon General Koop issued his landmark report on AIDS, stating "... education concerning AIDS must start at the lowest grade possible. . . .There is now no doubt that we need sex education in schools and that it must include information on heterosexual and homosexual relationships. The threat of AIDS should be sufficient to permit a sex education curriculum with a heavy emphasis on prevention of AIDS and other sexually transmitted diseases."

AIDS — WHAT DO ALASKANS KNOW?

- While most Alaskans are knowledgeable about the basic biomedical facts concerning AIDS and know that AIDS is transmitted through sex or AIDS-infected blood, many still harbor unwarranted fears about the spread of the disease through casual contact.
- Ninety-five percent of those responding to a recent survey know correctly that AIDS is a condition in which the body cannot fight off diseases. Ninety-eight percent know also correctly that AIDS is sexually transmitted, and an equal number know it can be transmitted by sharing a needle with a drug user who has AIDS.
- But Alaskans are much less knowledgeable about casual contacts and the spread of AIDS. Twenty-eight percent of those responding, for instance, believe *wrongly* that AIDS can be spread by sharing drinking glasses with someone with AIDS. An additional 18 percent are not sure. Fifteen percent believe *wrongly* that kissing can spread the disease, while an additional 18 percent are not sure. Sixteen percent believe *wrongly* that mosquitoes spread AIDS, while 16 percent are not sure.
- Many Alaskans also believe that somehow *donors* of blood are at risk for getting AIDS. Another significant misconception held by many is that a new vaccine has recently been developed for the treatment of AIDS.

AIDS — WHAT SHOULD ALASKANS DO NEXT?

- We must continue and strengthen our present Alaska AIDS program. We must stop considering only the number of AIDS cases and begin to focus on HIV infection. HIV infection is a far more serious problem; for every AIDS case, experts estimate that 50 more are infected with the virus and they are capable of infecting others.
- We must increase screening for HIV infection and counselling individuals who are infected or are members of high risk groups.
- We must implement the National Academy of Sciences and Surgeon General's recommendations to provide education concerning AIDS in our schools at the lowest grade possible.
- We must anticipate the future serious problems that will occur in health care financing and insure that Alaskans who develop AIDS and their families will have access to appropriate medical, health, and social services.
- We must insure that confidentiality is protected to assure the voluntary cooperation in the testing of Alaskans at risk for HIV infection and other communicable diseases.
- All physicians must become educated about AIDS. Accurate information is essential. Resource materials can be obtained from the ASMA office or the Alaska AIDS Program, Section of Epidemiology, Division of Public Health, DHSS.

Letters to the Editor

First Alaska ACP Chapter Meeting

Dear Editor:

The sun was shining in Sitka! Climatologically and psychologically and intellectually the first Alaska Regional Meeting of the American College of Physicians (ACP) was a brilliant success. Those of us from the "Lower 48"—ACP Regent Marvin Turck, M.D.; ACP Governor (Florida) Eugene Schiff, M.D.; ACP Health & Public Policy Department Director Deborah Prout, and I—basked in the hospitality of our Alaskan College family. Rarely has a two-day meeting packed in so much exciting new information. After Alaska ACP Governor Tom Wood, M.D. greeted us with a warm introduction, Marvin Turck, M.D. took the podium and delivered a series of provocative vignettes relating true stories of patients who had infectious diseases that challenged the audience. The participation was terrific. Then we were treated by Dr. Burton Janis from Anchorage to a discussion of a mechanism of action of a variety of antiviral agents. The morning concluded with another performance by Eugene Schiff, M.D. presenting an array of complications of cirrhosis and methods of management. It was a fascinating overview. I then had the pleasure of delivering my final "oration" on behalf of the ACP and expressed my delight at being able to be present at the first Alaska Regional Meeting.

The afternoon was the source of another "first." The first day of the "All-Alaska Logger Competition" was being held just a few hundred yards away. We all trooped over to watch this remarkable demonstration of prowess with axe and saw. It was a rare treat. The day was concluded with a cocktail party that featured chamber music with a trio of piano, viola and violin featuring selections from Beethoven. The finale was a remarkable voyage in a glass enclosed silent vessel to Silver Lake. I counted thirteen bald eagles along the route of the magnificent wooded inland waterway. The event was replete with a marvelous shipboard dinner that featured delicately baked fresh salmon.

I must say the range of extracurricular activities reflected the versatility of our Alaskan colleagues all the way from Beethoven chamber music to the logging contest. I challenge any other Region to top that.

The second day started with Marvin Turck, M.D. treating the audience to an update in genitourinary infections followed by Eugene Schiff, M.D. in a broad-based discussion of the hepatitities ranging from drug-induced all the way to the complexities of the viral hepatitis varieties.

A rare experience was presented by Brian McMahon, M.D., a Fellow of the College, who is in the Department of Medicine of the Alaska Native Medical Center in Anchorage. Dr. McMahon told of the astonishing Alaskan experience with hepatitis B. He had identified epidemiologic information concerning the incidence of hepatitis B in Native Alaskan populations that probably represents the most complete information available. He also reported on the effects of an extensive hepatitis B vaccine program. This was followed by an intriguing discussion by another ACP Fellow, John P. Middaugh, M.D., State Epidemiologist, Division of Public Health of the State of Alaska, who discussed the ethical dilemma of personal choice vs. public policy. He presented a variety of data in the arena of preventive medicine (i.e., AIDS, alcoholism, 3-wheeled ATV accidents, handguns, etc.) and cited the conundrum of "personal choice" vs. responsibility to the public. I was delighted to see John refer to the ACP *Ethics Manual* and our Public Policy Statement on AIDS as examples of enlightened documents that would assist working practitioners tread the delicate path between respecting the integrity of the patient confidentiality covenant and the need to protect the public against preventable intrusions into their health and safety. It was one of the most thoughtful, provocative and challenging discussions I had been privileged to hear.

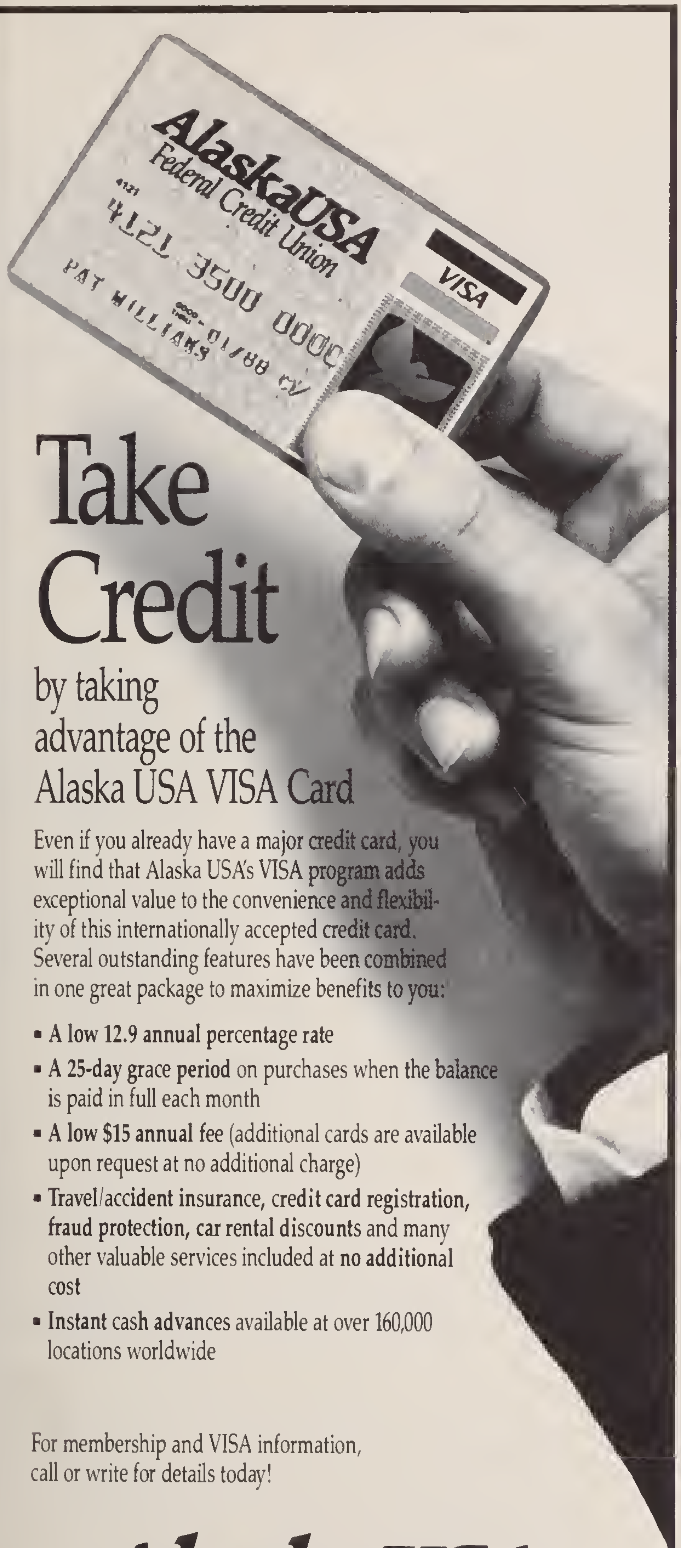
Then we saw the HEALTHSCOPE film featuring Dr. Timothy Quill of Rochester who dramatically presented the multifaceted problem of depression and anxiety. Then Miss Deborah Prout-Peterson, our Director of Department of Health & Public Policy, discussed ACP public policy operations and presented an update on a variety of important legislative issues.

It was the conclusion of a thoroughly delightful two days.

Governor Tom Wood, M.D., his wife Kathryn, and his team deserve a rousing "well-done." It was my final official act on behalf of ACP, and it was among the most memorable. I only hope that Tom and Kathryn have established a precedent that will be the start of a fine tradition of ACP Regional Meetings in Alaska.

The sun has never shown more brightly in Sitka (and to top it all off Guy German, a native Sitkan, won the speed climb in 14.08 seconds-just .06 off his 1985 record.)

Robert H. Moser, M.D., F.A.C.P.
Executive Vice-President
American College of Physicians
Philadelphia, PA



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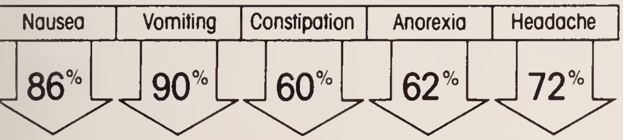

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Anticholinergic Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus

Hematologic. Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal. Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue

Endocrine Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion

Other Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling

Overdosage: Immediately hospitalize patient suspected of having taken an overdose Treatment is symptomatic and supportive I V administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning See complete product information for manifestation and treatment

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THE COMMITMENT CONTINUES

Which Way to Turn?
Inuit Culture, Alcohol, and Change

by Carl M. Hild, M.S.[1]

ABSTRACT

The indigenous people of the North American Arctic have had their culture highly modified by the migration of the Western European Culture around the globe. One of the primary signs of this impact has been in the use of alcohol. A review of cross-cultural studies and methods utilized in alcohol research led to the drafting of this presentation on the history, past problems, need to improve, and the new structures in place for improvement in the area of alcohol research and treatments for its abuse in circumpolar regions.

Trans-cultural techniques should be eliminated while cross-cultural programs are expanded; broader support of traditional structures and methods to strengthen individuals and communities; and more flexibility from western European care-givers; all are needed to enable the Inuit to reorient themselves in their rapidly changing world.

ALCOHOL IN THE ARCTIC

The indigenous people of the North American Arctic, the Eskimo as most of the world calls them, the Inuit as they call themselves, have had their culture highly modified by the influx of western European cultural values. One of the primary signs of this impact has been in the use of alcohol. Before its introduction by white or non-Native explorers and frontiersmen, the Arctic people did not have their own intoxicating drink. Once introduced, its use was described by various white authors (1–5) as having been immediately accepted (6), and then rapidly progressing to uncontrolled use. Aspects of this problem unfortunately continue to prevail even today (7–29).

The Inuit, however, are not alone with respect to their alcohol-related problems. Among Arctic or Circumpolar countries, alcohol abuse is a severe problem both for Natives and non-Natives alike. Research has shown that Arctic areas generally seem to have a greater problem than more southern areas (30–32). Russia, for example, has recently declared war on alcohol. Iceland has had serious problems. The Nordic countries have had a long-standing problem with alcohol. The residents of northern Canada and the United States also share a common problem with alcohol. Even organized, western cultural military bases and scientific research stations are not free from tragic alcohol related incidents, including killings resulting from the drinking of "home brew."

The realization that Arctic areas show a high prevalence of alcohol abuse has led researchers to look for common causes. The extent to which the cause is primarily attributable to either cultural, climatic or geographical factors is not known. Clearly an interaction exists. Nevertheless, with respect to the Inuit, their physical environment, except for their very recent exposure to western ways and technology, has remained relatively constant. Light or dark, still or storming, hot or cold, the environment has not changed. It would thus appear that for the Inuit, factors other than the physical environment may contribute more to their abusive drinking patterns. This leaves one to determine the relative contributions that cultural factors play in drinking behaviors.

It should be noted that alcohol abuse in Alaska is not only within the domain of the Eskimo and other Native groups; high consumption rates exist for non-Natives, whether in Arctic or sub-Arctic environments. The situation has been described as follows:

> Alaska was, in short, just the locus of points enclosed by a certain line on the map, and about the only things "Alaskan" shared by a Croatian gold-dredge mechanic in Fairbanks, the Tlingit fisherman in Hoonah, a teaching nun on the Lower Yukon, an Eskimo hunter in the Arctic, an Irish wheel-box oiler in Chitina, and a Coast guard radioman based in Kodiak, were a desperate struggle with the weather, the tenuousness of ANY ties they might have with ANYBODY outside Alaska, and more likely than not, a love affair with demon rum (33).

THE INUIT AND ALCOHOL

In Alaska, most NON-NATIVES do not perceive THEIR culture as having an alcohol problem. Alcohol is accepted as a traditional part of their society. They view the Natives as the ones with the alcohol problem and attribute the cause, in large part, to the relative newness of alcohol to Native cultures, and to the weakness of Native people to resist its influence.

Alcohol has been with western civilized man for thousands of years. Egyptians wrote in hieroglyphics of

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the hazards of too much beer. England in the 1700's had an excess of grain. Rather than let it spoil, it was made into gin and the phrase "Penny Gin" was born. Symptoms of what is now identified as Fetal Alcohol Syndrome were first reported by English physicians during these times when pregnant women had plenty of "Penny Gin." Social disruption and disagreeable behavior abounded in those days. In time, in the cultures of the world that have had alcohol, the people began to learn how to use alcohol and to behave in acceptable ways more often than not; there was less social discord.

The effects of alcohol are not absolute, but what a particular culture or community is willing to accept as appropriate or inappropriate drunken behavior (6). Reading historical accounts of alcohol's introduction to the Natives of North America, it seems that when it was first offered, often by diplomats and governmental representatives, the Native leaders did not like it (6).

The Inuit, in contrast, did seem to enjoy the taste and actively sought out more (1–3, 11, 34). The rather rapid acceptance of alcohol occurred because the Inuit, who are excellent mimes and use their abilities to copy animal behaviors in order to approach game animals, applied the same basic techniques to acquiring drinking behavior — they emulated the patterns of drinking observed among the early white visitors to their land. Behavior was observed, copied, practiced, and tried again to match the original. "Imitation is the sincerest form of flattery," and since the Inuit were so impressed by the technology of the western society, they copied many of the new gadgets and the behaviors of the owners.

Looking at Native drinking patterns today, there is clear evidence about who first introduced them to drinking. The first westerners the Inuit were exposed to were the fur traders, frontiersmen, and whalers. Never did a more raucous group of binge-drinking, womanizing, horde of social misfits have the prime opportunity to introduce a virgin and distant culture to the finer points of the western European ways of consuming alcoholic beverages — drink until the barrel is dry! "Yo ho ho and a bottle of rum." The whaling accounts are full of stories of drinking in the far north (1-3).

Surprisingly, the missionaries also had a role in the Inuit's rapid acceptance of alcohol. The missionaries, the counterparts to the early explorers and exploiters, were the various Christian evangelists seeking new souls. They were so businesslike that, to save money, they agreed among themselves to divide Alaska like a pie, and each sect could go after just one piece. No competition! This focus of all resources on smaller areas led to intensive bouts of eliminating Native "heathen" beliefs. The Natives, in their confusion, asked: Who is this white man really? First he visited and takes some of our tools and clothing. Then he wants furs, baleen, ivory and our women and gives us drink for them. Then he wants us to give up the drink he has given us, love his spirits, learn his stories, and forget our own stories and beliefs.

The missionaries told of how the loving white shaman, Jesus, would forgive them if they sinned or were weak. This situation contributed to an acceptance of binge-drinking since one could be a good church-going Christian most of the year, but if the drinking occasion occurred, it was all right in the eyes of the white shaman as long as forgiveness was requested. The missionaries followed their own rules and only theirs in relating to the Natives. They showed no interest in appreciating anything about the Native culture except the Inuit conversion to their "God." " 'Obey God and Jesus; don't steal; don't talk lies.' This struck the members of the group as rather inane, particularly since stealing in the aboriginal culture was virtually unknown and no self-respecting individual would think of telling a falsehood." (35)

Along with the pressure to give up their traditional spiritualism values and accept Jesus, there was also pressure to learn a new language and live in a non-nomadic way. White contact in many areas of the Arctic is less than 100 years old. Regular, year-round white contact in some areas has only occurred since 1950. Some villages received television for the first time in the mid-1980s. Alcohol has a relatively short history in the Inuit culture. Their initial exposure to drinking was based on their imitation of the binge-drinking patterns practiced by the whalers and fur traders who visited their communities, bringing alcohol with them. After this introduction to alcohol the missionaries came, who preached, and who attempted to enforce abstinence.

Historically the Inuit culture had "time-out" periods (6), a phenomenon also noted among American Indians (20, 36). These are times when community rules are relaxed. This practice, within a hunting society, lends itself perfectly to the occasional style of drinking. In more recent times the "boom/bust" cycle of economic activity in the Arctic has been a factor that reinforces these cycles and binge-drinking behavior. Furthermore, access to these distant rural villages, via air transportation or seasonal boat shipments, also reinforces binge-drinking.

An important issue that has resulted from observation of binge-drinking is whether there is a high incidence of "alcoholism" or "problem drinking" among the Inuit. Some (17) have called it "alcoholism," while others (26), describe it as a form of an "alcohol-related problem." The underlying issue, however, is the western philosophical approach to alcoholism, in which the disease concept of alcoholism is embedded, and which defines all forms of alcohol-related problems as alcoholism. The utility of this model has to come into question when the drinking behavior of other cultures is examined (6).

ALCOHOL TERMINOLOGY AND NATIVE DRINKING

An important issue in the study of alcohol and its effects is the nature of the vocabulary used to describe drinking-related problems. Labeling groups of Native people or Native communities as "alcoholic" (17), for example, does not make sense when the term is defined in context of western concepts of drinking–related problems. Terms such as "alcohol abuse" can potentially result in one of two types of diagnosis — "alcoholism" or "alcohol abuse." Drinking behavior, as any other form of behavior, can be considered a learned behavior and thus needs to be labeled in terms of the context in which the behavior occurs (6).

> "Drinking is a learned 'way' of a named group of people. For that group, its own way is the way, the appropriate, proper, natural, and decent way to drink alcoholic beverages. People who drink in a significantly different manner from others in their group are likely to be called alcoholics. Their deviant drinking behaviors will be regarded as symptoms for the existence of alcoholism (37)."

In addressing the problem of what terms to use when describing or discussing Inuit drinking behavior, the term "alcoholism" will mean the physical and social addiction to alcohol. It is analogous to the "gamma" alcoholic (38). The phrase "alcohol abuse" will be used nonspecifically, representing drinking behavior that extends beyond what the local community adopts as acceptable drinking. Excessive or "deviant" drinking, or "alcohol abuse" represents drinking behavior that extends beyond these norms. In some instances even the consumption of alcohol to a point where it may negatively affect the individual's physical, social or mental well–being may not be considered alcohol abuse. Thus the tolerance for drinking–related problems differs greatly within Native communities, and in general contrasts sharply with non–Native standards.

When Native individuals are labeled as "alcoholic," in the traditional sense of the disease concept, they react in terms of their cultural values, which involves a significant degree of fatalism. The reaction may be expressed as: "I have a disease. I will continue doing what I have done in the past as long as I am able. I will not complain nor change, but accept this as my fate."

This fatalistic attitude may explain why alcoholism treatment programs show a high failure rate (39). Another reason for failure is that when a diagnosis of alcoholism is made, the "patients" do not fit the expectation of the western notion of an alcoholic; nor do the Natives themselves adopt the appropriate role of an "alcoholic." Indeed, many of these people may not be alcoholics at all, but are "social abusers." The abuse is a manifestation of an offense against the community, rather than a personal difficulty. People with drinking–related problems generally come to the attention of authorities primarily when the community cannot control their behavior, and not with the realization that they may be developing a "drinking problem." The "patient" would thus approach "treatment" from a different set of expectations than those of the treatment agency. The drinker wants to know how to redeem one's self within the community, and the treatment counselor wants to confront the patient with his or her "alcoholism" condition.

Alcohol abuse, nevertheless, does exist, and to a large extent. It is important to note that even if there is an overlap between what western practitioners call alcohol abuse or alcoholism, Native communities have their own standards by which they define the problems associated with the consumption of alcohol. If interventions are made, or alcohol studies undertaken, then they have to account for the Native cultural perspective.

Although alcohol–related problems are a national problem (40), such problems, when they occur in small (population 10–500), isolated, communities, represent an extremely dramatic problem. The problems resulting from abusive drinking consist of a wide range of adverse effects, including death, as recently illustrated by the demise of four young people from a small Native community who drank methyl alcohol. Their deaths not only had a significant emotional impact on the community, but contributed to a significant increase in the per–capita alcohol–related death statistics in the community, and in the local Borough as a whole. If these deaths represent, for example, approximately one percent of the community's population, an equivalent amount of deaths in Anchorage, for example, would amount to 2,500 deaths overnight due to one cause. If a proportionate amount of deaths were to occur in New York City, then approximately 80,000 sudden alcohol related deaths would occur. It can only be speculated how the public would react to such a catastrophic occurrence.

Yet in rural Alaska such high death rates are not uncommon, but statistics about types and levels of alcohol–related health and social impacts evoke only momentary reactions. Such statistics are not generally gathered by experts in local communities. "Outside" consultants are usually hired to gather and interpret them, and write reports directed against alcohol abuse. Oftentimes these proposals are not written to help the community itself understand and address their problem, but are directed at influencing others to react to the problem. When this occurs, and if the community is successful in obtaining funding, then they often have to recruit "outside" people to run the programs. These individuals may have an understanding of alcoholism, but have no understanding of the way of life in the community, nor of how their knowledge of alcoholism applies to the community in which they find themselves.

A similar problem exists with respect to conducting alcohol research in Native communities. Studies stand a better chance of success with respect to helping the community when they are acceptable to the people they affect, and when the results are written in a manner that is understandable to the local community (41, 42). Studies fail to gain support from local communities when they tend to be initiated by outsiders who are interested in collecting data for their own needs, rather than to work WITH the community to help address the needs of that community. This problem is compounded further when the researcher, in a community of mixed ethnic groups, chooses to focus only on the Native as the group with a drinking problem (13, 17–19). Despite the fact that some of this work (13, 18, 19) was repudiated by reputable authorities (14, 15, 25, 26, 28, 42–44), the authors continued to publish their findings as gospel and continued to misrepresent the facts with respect to drinking behavior in the community (17). More interesting, these researchers dismiss the criticisms of the reviewers as professional envy, rather than as objective commentary. The net impact of this situation was that it precluded researchers, because all researchers were no longer trusted to be fair and honest in their dealings with Native people, from entering into study in that community.

When the community relented to allow research to ascertain the prevalence of substance use, a firm condition was established that precluded disclosing the findings by the researchers. When this condition was met (45, 46), the researcher described the findings pertaining to the community in a manner that precluded it from being identified as a separate community, and wrote a special report just for the people of that community that allowed them to control the findings as they saw proper. After the research was reviewed, the school board chose to go public with the findings about drug use in the junior and senior high schools; a decision which was theirs to make, not one the researchers made for them.

When researchers become myopic, they look at things only through their particular brand of "rose colored glasses." They read literature not applicable to Native communities, they write in terms of their discipline's jargon, and fail to communicate with the consumers of their research — the ones who "paid the bill." If change is to occur, multidisciplinary approaches are needed which take cross-cultural issues into account. One author developed a model for Alaskan alcoholism and drug abuse treatment service delivery BUT NEVER ONCE mentions Native people or cross-cultural issues in a state where 15 percent of the population is Native, and where a large percent is from the Orient.

Researchers come and go. They arrive, "do their thing," leave, and publish their results in journals that are never read by the people they studied. Researchers are particularly disliked in Native communities when they come, observe, interpret certain behaviors as they see them, without ever truly understanding the basis of those behaviors. They interpret the events out of context, and from a different cultural world view. Native people believe that such researchers should spend an extended period of time with them to learn about them and their cultural practices. After five years of friendship a Barrow Inuit said jokingly when asked if he had ever been interviewed by researchers; "No way! Those researchers would have to beat it out of me." Then he turned serious; "No, they should learn on their own. Let the Arctic teach them." Another Inuit friend said after an unexpected interview by a researcher; "How dare they just come into my house and ask questions. They don't even know me!" These statements need no elaboration with respect to how they convey the Inuit feelings on being the "objects" of research by those perceived as being "outside" researchers.

When researchers enter a new environment they tend to practice problem deflation, that is, understate a problem, and not view issues in the same context as the residents (47). They also tend to bring lots of their own "cultural baggage" with them (14, 26, 47-50). Margaret Mead believed that the single most important preparation for fieldwork was to be sure you completely understood your own culture before you went into someone else's. Many researchers' baggage consists of a suitcase full of arrogance.

> We in the West are convinced that we have a corner on reality — a pipeline to God — and that the other realities are simply superstitions or distortions brought about by inferior or less developed systems of thought. This gives us a "right to free them from ignorance and make them like us." The dazzling success of our technology, as well as our understanding of the physical world, has blinded Europeans and Americans alike to the complexities of their own lives and given them a false sense of superiority over those who have not evolved their mechanical extensions to the same degree. Science is our new religion, and in many instances, like old religions, it has served man well up to a point (51).

Since western culture has been unable to find the right means to deal with alcohol abuse in its own setting, perhaps it should hold off confronting a different culture with its dysfunctional solutions for a problem it does not understand, even though the issue was initiated by transmitting alcohol from the western culture to the Inuit culture.

TRANSLATIONS

Dealing across cultural barriers is not just an issue of translation of words. In translating Inupiaq to English, or the other way around, the words do not always match. There is no Inupiaq word and therefore no concept of "luck" (52). There are dozens of words in Inupiaq for types of snow.

Inupiaq words concerning time are very limited, since time has a very different meaning in the Inuit culture (53, 54). Words for large numbers and units of measure do not have relevant parallels. Nutritionists are plagued with the lack of an Inuit definition of a "meal." Alcohol researchers need to be aware of the lack of a common definition of a "drink." Therefore, asking questions in alcohol research about "How many drinks do you have in a . . . (given time period)?" has little or no value. Moreover, Inuit do not like hypothetical or imaginary situations (55). Therefore, "what if" questions are poor selections.

Basic "yes/no" questions, when translated from English to Inupiaq, will often result in the reverse answer being given, resulting in the researcher wrongly concluding that appropriate answers were obtained. Inuit children in school confound teachers by using the traditional non-verbal response to "yes/no." They are often chastised for either not answering or not speaking-up. The children become confused as they did answer and now have been embarrassed in front of the class.

Inupiaq words and phrases have high-context, while the English language is one of low-context. A high-context communication or message is one in which most of the information is either in the physical context or internalized in the person, while very little is in the coded, explicit, transmitted part of the message. A low-context communication is just the opposite, i.e., the mass of the information is vested in the explicit code (51).

HIGH-CONTEXT

> My family was camped in the summer by the sea. They were hungry so my father took his kayak and went off to find food. While out on the water a fog developed. The fog was so thick and it spread over the water. Soon my father could not see and tried to turn out of the fog, but he was unable to. He waited, but the fog did not blow away. He was sad for he could not get the food to his family. He floated and then cried to the water to send him a sign on how to return home. Suddenly a large killer whale appeared. He said; "Oh killer whale, I am a hunter like you; I have food for my family. I will do you no harm. I am confused in all this strange fog. Please help me find my way." The whale circled around the kayak a few times and then swam off staying on the surface. He paddled hard behind and was just able to see the large black fin. He trailed the whale until he was almost exhausted and then he lost sight of the fin. Off through the fog he could see his camp on the beach. He thanked the spirit of the whale and said that he would never harm any killer whale.*

LOW-CONTEXT

> The Inuit have survived many centuries in the Arctic. Then the environment changed one day with the white man's arrival and the introduction of alcohol, which had a negative impact on the traditional way of life. There was confusion and people lost sight of the things that were important to them. All seemed lost, and rather than take definitive action, the Inuit people waited for the white man and his technology to provide the solutions to their problems. There was not help for the problems brought by alcohol. In the turmoil and change the Inuit lost hope and self-esteem. Finally an attempt to renew the traditional skills and values was tried in order to help the generations of the future. The traditional approach was to request help from the spiritual resources that have been with the Inuit people for all of their history. Through this belief, acceptance of all that life brings and subsequent definitive action, the Inuit are able, after much effort, to return to their true culture and to restore the traditional strength and outlook on life to its people, and to assure themselves a positive future.

> "In cultures in which people are deeply involved with each other, and cultures such as the American Indian, in which information is widely shared — what we will call high-context cultures — simple messages are likely to be overwhelmed by mechanical systems and lose their integrity (51)."
>
> "In general, high-context cultures — those which call for considerable programming of individuals to each other — have greater mass and are therefore more predictable, if, and only if, one is familiar with the system. On the other hand, to the observer who doesn't know there is another system, a strange high-context culture can be completely mystifying, although he may not know or accept the fact that he is mystified (51)."

High-context communications imply prediction of culturally-related activities. Since high-context communications require time for the culture to evolve solutions to new problems, the Native community, because of the relative newness of alcohol within their culture, has had insufficient time to derive acceptable cultural solutions. Since there are not traditional solutions, the next generation may have to deal with the problems related to alcohol. With the culture in a state of change and reeling from the rapid, radical impact of western culture, traditional style solutions may be a long time in coming. Among the Inuit, the youth and the elders are currently struggling to cope with the adverse impacts of alcohol and drugs. However, working culturally acceptable solutions to these problems may not evolve until the next generation or possibly not even until the third generation.

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*Hughey, Betty Nukapigak. Story, with permission to apply it for this use. Personal Contact, 1986.

With the new Inuit cultural norms about alcohol will come new attitudes to deal with associated problems. One of the traditional Inuit cultural ways, for example, that may be used differently to deal with alcohol in the community is the concept of "shame." This Inuit practice avoids inducing confrontation and any resulting anger from the one who deviates from community norms.

The concept of shame involves not confronting people with any misgivings, nor evoking retribution, nor showing intolerance toward behaviors which would be judged "deviant" in the western culture. Rather than "condemn" or confront the person, the behavior is tolerated, and it is believed that through this toleration of poor behavior, and either gentle teasing or shunning, that the offending person will eventually correct his or her ways and return to the community norms. If the person's behavior became extreme, then the traditional practice called for the community to banish or completely shun the individual in order to preserve the community's integrity. Banning is no longer effective because of the transition from a nomadic, hunting society to a larger, permanent diverse community with more of a cash based economy.

The concept of shame, however, still prevails but has lost its traditional effectiveness. Since shame is not working as it used to, it is interpreted by western practitioners as complete tolerance and therefore acceptance of any drinking behavior, and as "enabling" the alcohol abuser. Western observers should not be quick to label or judge cultural practices out of the context in which they occur. The question for the Inuit people is how to utilize this cultural practice more effectively once again, and how to use it to deal with the new problem of alcohol abuse.

Another attitude within the Inuit culture is to not show assertiveness. An Inuit friend, for example, said that when he was growing up he was told not to try to be "it" not to try to be the best at hunting or at school. If he was the best he would soon find out from others, but it was not his task to seek to be "it." An Inuit hunter does not go out to hunt Eider ducks. He goes out for food. If he is presented with ducks, he shoots ducks and has food. With this attitude no self-respecting Inuit can say someone else has a drinking problem without the unstated assertion that "I know how to deal with alcohol." Therefore little or nothing is said to the alcohol abuser. It is also totally inappropriate to criticize anyone for any reason after their death, even if their death was completely due to alcohol abuse.

In summary, what is important is the realization that the Inuit and western cultures are very different. Although many behaviors may look similar, they may nevertheless occur for different reasons, that is, based on different world views. The question for the non-Natives working and living with the Inuit is: How does one culture deal with another culture whose traditions and day-to-day practices are dramatically different from those that are predominant in the American or western culture?

STUDIES AND TREATMENT OF ALCOHOL ABUSE

When one culture's methods of studying and treating alcohol abuse are applied to another culture, the process is called cross-cultural research or treatment. However, the adoption of one culture's methods to another is not only inappropriate, but does not constitute true cross-cultural practices; it represents trans-cultural practices (26). As such, the traditional western tools for dealing with alcohol abuse (and mental health problems) are, in large part, inappropriate for Native cultures. For example, many of the psychological tests used in Alaska to assess the mental status of Alaskan Natives are inappropriate (56 - 58). Some tests that have been reported as being effective when used cross-culturally are the Rorschach and the Thematic Apperception Test (TAT), but these are biased by their user's interpretation (56 - 58). With respect to this biased interpretation in using the Minnesota Multiphasic Personality Inventory in a cross-cultural setting, it has been noted that: "It is largely because of this particular issue that criteria-based tests such as the Minnesota Multiphasic Personality Inventory (MMPI) are thought to be inappropriate for intercultural use, whereas construct-related tests are generally considered to be more acceptable (59)."

"Story Telling" is a traditional Inuit oral practice or way of communicating. This could be used as a tool (57). Other western tools might also work. The Alcohol Dependence Scale (ADS) (60), for example, could be used if there were local norms from which standardized scores could be developed. If the ADS could be translated into Native languages, tested for reliability and validity, and then administered to a large enough sample, local norms could then be developed to assess the degree to which the drinking behavior of any Native individual is consistent with or departs from normative community practices.

This procedure, however, is not as good as developing a measure designed specifically for Native people, one based on their language systems and cultural values. If assessment procedures or techniques are to be used, then one area for future research is the development of a Native culture–bound alcohol assessment scale. "Yet the peculiar social/cultural influences that contribute to the development of alcoholism, or its successful treatment, in these populations . . . are so little understood that it is difficult to establish priorities for service or research efforts that might ameliorate the problem (61)."

Treatment for alcohol problems in the United States has chiefly been centered on older, white males. The popular treatment mode for such individuals tends to be Alcoholics Anonymous (AA). There is considerable debate, however, whether AA is a method of treatment, a philosophy of treatment, or a religious belief. AA, nevertheless, best serves people who have decided that they want to live without alcohol, may of whom Eric Hoffer would call "True Believers" (62). These are not usually the alcohol abusers of the Arctic; the young, single, Native men and women in rural communities. Moreover, AA is based on a Protestant–Christian ethic, one that is not totally consistent with the value system of the Inuit, many of whom are Christians. AA groups are not numerous in Alaska and are hard to start and maintain in outlying Native communities of less than 500 people. If AA is experienced as unsuccessful, then there is resistance for others to try it, and in the absence of other treatment alternatives, many feel that there is no help at all.

Added to this is the problem that to get help means leaving one's home environment to go to a residential program many miles away. Even when an intervention is successful, and the person does not want to drink again, when he or she returns to their original drinking environment there is significant social pressure to drink, and oftentimes there is no support for continuing to abstain. Small community social participation and belonging is an extreme force. Resistance to abstain is therefore shortly eroded and drinking often resumes soon after the return home.

The best way to illustrate an example of well–intentioned but ill–conceived research and treatment among Alaskan Natives by non–Natives is to tell a story about what occurred in Barrow, Alaska. A non–Native alcohol counselor decided that the source of much of the alcohol abuse in Barrow was due to unresolved grief. He attempted to construct a program around the idea. Before long he left the community, which is not atypical of health–care workers who work in Barrow or other rural areas of Alaska. Another white counselor decided that the grief idea had merit, and he raised $30,000 to bring Elisabeth Kubler–Ross, a world renowned expert on death and dying, to Barrow to conduct workshops on grief. As with the 1980 study, described previously, the workshop did not have broad community support. Both were seen by the community as being led by "white knights," and after the exercises were over little had changed, the "white knights" had left, and the money and effort spent on both of these projects were essentially wasted. Money, time, and energy were spent on ideas that did not fit the community's needs nor personality.

When ideas about drinking within the Native community, or in any community outside one's own immediate culture, are conceived in ethnocentric terms, there is every expectation that the notions conceived are forced interpretations to account for specific events, such as drinking. While such ideas may be initially responded to, the fact that they are not based on the culture's reasons for doing the behaviors ultimately leads to the failure of such notions.

In contrast to ethnocentric thinking, observation of events within the Native community can lead to a better understanding of events related to drinking (22, 27, 63). Some, for example, refer to the fact that forty–year–old Native people changed behavior and ceased drinking spontaneously. This is about the time a transition takes place from merely being an active member of the community to becoming an community leader, a step in becoming a traditionally respected "elder." This pattern is sufficiently consistent to suggest that it may be a form of self–regulation in the Native community for individuals who are becoming of proper age and proper status. They are not expected to drink in excess when they hold responsible positions within the local community.

Numerous researchers (41, 42, 64) state the importance of community acceptance of the problem and of their own solution. Part of the final report (65) given at the 1986 Inuit Circumpolar Conference stated that it is unrealistic for non–Natives to expect and require that Native groups use western cultural structures to deal with traditional issues. Anyone of western cultural background who works in a standard alcohol treatment program cannot expect and require Native individuals and groups to utilize the program in the same manner as westerners. Similar assumptions have been made regarding other indigenous Native groups. Some (60), for example, see "Indian Alcoholism Treatment Programs as Flawed Rites of Passage," and others (66) describe Indian drinking patterns as a form of protest against the western system.

A realization by Native communities that self–help, in terms of the community's values and traditions, has led to a revival of some traditional ways. The emergence of the "Spirit Movement" within Alaskan Native communities represents traditionally inspired programs that are oriented toward problem-solving (67, 68). An important component of the Spirit Movement is traditional dancing, a form of "Story Telling," that facilitates self-identification with, and perpetuation of, Inuit cultural values. Not everyone can be a respected dancer. In order to assume this role one has to go through a rigorous mental, physical, and spiritual transition.

A most intriguing concept is "Spirit Dance Initiations." Workers in Galena, Alaska, have stated an interest in psycho-rhythmic drumming (69, 70). This would entail establishing complete rites of passage programs. Not only would an individual go through a rigorous mental, physical and spiritual transition; the family would be intimately involved as well as a large portion of the community. All would invest energy for the rite, as well as continued support for the initiate after the program. Perhaps this is an area to be supported by grants to establish baseline data and to see if communities would accept such responsibility. Data collected to date seems very positive in regards to the initial and continued success rate of those who complete the process.

> "Karl Menninger who sees in peyote 'A better antidote to alcohol than anything the missionaries, the white man, the American Medical Association, and the Public Health Service have come up with.' . . . Traditional or syncretistic cult movements of Nativistic character . . . [are] effective therapy for alcoholism (70)."

The Spirit Movement, however, can only be initiated when a community itself seeks to take the responsibility to ensure that the cultural values are preserved and transmitted to the next generation. If the community is successful in the endeavor, then it may possibly replace the western physical or social health professional since the problems would then be responded to by the Native community itself. The role of the western health practitioner would then change from direct service provider, to assisting the community to further its self-help efforts. Perhaps these are areas that can be supported by agencies through grants to provide funding not for the agency to provide services, but to assist the growth of self-help movements.

SUPPORT FOR SELF-HELP

Not so long ago there was an old shaman's tale that passed orally into the 20th century. It said that one day people of a different color would come to the home of the Inuit and would take care of them. In the late 19th century missionaries arrived, and said that the Inuit beliefs were bad; believe in the loving white shaman, Jesus, and "God will take care of you." Then came the government representatives who, through the Bureau of Indian Affairs, built schools and told the Inuit that white teachers had stories that were better than the old traditional tales; learn these new stories and we will educate and take care of training your children. The Public Health Service, via the Indian Health Service's doctors and nurses, arrived and taught that the traditional health practices were not good; the Inuit should come to the new clinic for their health care.

The white man's being responsible for the Inuit met the shaman's tale and also fulfilled the westerner's self-perceived role of rescuer in dealing with the concept of "the white man's burden." The old shaman's story had come true. The Inuit no longer had reason to do things on their own until they realized what they were losing. They had lost their spirit; they were losing their Inuit self. The pendulum now seems to be swinging in the other direction. "After having indulged to exhaustion into the charms of modern times they are coming back, slowly, to a serene appraisal of the old traditional values" (71).

Self-help is not without its price. The Inuit are perplexed in that western culture requires them to play only by the western culture's rules; e.g., legal action, parliamentary procedure, by-laws, and corporations. These are structures that do not work efficiently in small rural communities where English is new and there has not been, until recently, low-context written language of any kind. When legal matters arise, for example, a small village must retain a lawyer who not only lives far away, but who contracts for payment in cash — not in trade. Western professionals need to learn Native cultural practices, and attempt to work as much as possible within this framework. For example, grants-in-aid are accompanied by requirements for extensive "paper trails" to account for expenditures and document achievements. These grants often specify how the money is to be spent; however, the specified procedures are often at variance with the needs and desires of the Native community, and in the way in which they seek solutions to their problems. Perhaps it would be more advantageous to permit the Native group to use the money in ways they deem appropriate, providing that they are able to justify its expenditure. This will require administrators who know both cultural systems and can assure their co-workers that the programs are working as planned.

Native people have also been sold a false bill of goods by the western educational system. Schools prepare children for adulthood. Native children are prepared for western urban adulthood. College is portrayed as the "big reward." When Native children reach for it, most of them fall short. These false expectations and sometimes unrealistic, unachievable goals, have helped to establish a sense of frustration among Native youth, and this has contributed to setting some on the path of alcohol abuse. Schools should prepare Native children for Native adulthood. This still means that modern education needs to be available to provide opportunities for those who wish to become Inuit doctors, lawyers, or corporate chiefs. If students are to leave the community for further education, then they need to learn about living away from home and in a very different world. They need to learn about western competition rather than Inuit cooperation. Students also need to know about their own community, how their community works, and how they can work within their community to achieve change.

> If there is to be any noticeable change in the direction of less heavy drinking in village life then there must be marked change in the demands made of the people to be other than what they are. Is it not time for the white, dominant society to take some of the blows of introspection about our directions and demands? Can we of the white community not look at ourselves with a critical eye to the values we impose or imply? Have we enough inherent security to allow Native solutions to Native problems, or must we insist on our solutions to what we think are their problems? Increasing weight and responsibility should be put on those of us in the white community to expand our values to include a sincere appreciation for the beauty of the ethnic diversity which the Natives of Alaska have devised and maintained (12).

Support from the western cultural structure is appearing. Self-government, Native sovereignty, and home rule are in place in some areas. The enabling legislation for the national Arctic Research Commission provides that an indigenous resident be a member of the Commission. Local option laws for determining the availability of alcohol in each community are in place in Alaska. Changes are continuing to occur, and perhaps the most important struggle facing the Native people is working together to solve common problems; the key concern being the regaining of the responsibility for dealing with their own problems. A responsibility that was falsely and unfairly removed from them by the western culture in an attempt to "help."

Several recent actions by various national and international boards or commissions reflect a greater sensitivity to Native issues and a shift in some of the social responsibilities.

> It . . . [is] crucial to focus research on social and cultural conditions in order to reduce such behaviorally based pathologies as alcoholism, suicide, drug abuse, and violence. . . . Without question, one of the greatest challenges in the development of the Arctic is the well-being of Native peoples. High rates of suicide, homicide, accidents, alcoholism, abuse, and unemployment among Native groups are all salient indicators of stressed social well-being. . . . Most important, we need to understand how people in the face of pervasive social and cultural change retain a sense of control over their lives and an ability to cope with the changes they confront (72).

The Interagency Arctic Research Policy Committee has stated that in order to reduce adverse impacts on Native culture, studies should be done on the "Long-range health, behavioral, and social effects of development on Native cultures, including socioeconomic impact studies, social problems, and substance abuse" (73).

The Inuit Circumpolar Conference has established an International Arctic Policy. At a meeting in 1983, the Inuit Elders passed a resolution (E-02-83) that identified the "Inuit as a unique people with sovereign authority within the Inuit lands are to govern themselves and exercise traditional methods of control relating to the banning of alcohol and drug substances locally."

The American Public Health Association's National Arctic Health Science Policy recommends guidelines for the conduct of research in the Arctic. It states that:

> Communities should be involved in research only after they have been fully informed of the nature and type of work planned and consented to it. Procedures that have been developed by local communities or institutions involved in health research should be followed and appropriate approvals should be obtained. The funding agency must have written assurance that such approval has been granted. The research project must provide for reporting back to the community in a language and style that is easily understood by the lay person. Funding for research should include a scientist's visit to the community to present the results of the project and to solicit comments and questions from the community. In addition, a mechanism should be implemented to ensure that recommendations made as a result of the research are reviewed and brought to the attention of the agencies capable of and responsible for implementation (74).

WHICH WAY TO TURN?

With the new attitudes toward self-determination in the Native community, and changes in the way support is to be provide by "outside" agencies, come new questions.

For the Native: Which way to turn? Will it be toward acculturation, allowing care and responsibility to continue to be placed on the western culture as the shaman's story foretold? Or will it be toward reidentifying the Inuit community's and traditional personal strengths, looking inward to deal with the rapidly changing world around them, and accepting the responsibilities of taking action within the existing structures.

For the non-Native: Which way to turn? Will it be toward continuing to insist on acculturation and dealing only in a one-way, trans-cultural effort, using Natives who know both worlds as "culture brokers" to sway other Natives to westernize? Or will it be toward developing realistic cross-cultural programs with a two-way effort and freedoms for the minority culture to exist, facilitating self-determination through grants for traditional or locally supported approaches to health and substance abuse programs.

CONCLUSION

When the wolf, the hunter of the tundra, knows that the caribou have moved too far or are too few, he will go to the ocean. You may see that wolf eating grass or drinking from the ocean. You will know that it is not healthy or strong. The wolf may die or it may change its form and become a killer whale and hunt the ocean. He will hunt for long times. When the killer whale, the hunter of the ocean, knows that the fish and seals have moved too far away or are too few, he will go to the beach. The killer whale may die or it may change its form and become a wolf and hunt the tundra.**

We, from the western culture, should understand and allow the Native to change his form, as the spirit within him needs to stay strong. We all need to listen with two sets of ears and understand with two hearts. Which way will we turn?

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QUOTE FOR THE QUARTER

Tobacco smoking is a form of drug dependence, and the modern cigarette is a highly efficient device for self-administering the drug nicotine. By inhaling, the smoker can get nicotine to his brain more rapidly than the heroin addict can get a "buzz" when he shoots heroin to a vein. It takes only 7 sec for nicotine absorbed through the lungs to reach the brain, compared with the 14 sec it takes for blood to flow from arm to brain. Furthermore, the smoker gets a "shot" of nicotine after each inhaled puff. At 10 puffs per cigarette, the pack-a-day smoker gets more than 70,000 nicotine shots to his brain in a year. It is hardly surprising that cigarette smoking is so addictive.

Editorial Note: The average Canadian smoker consumes 30 cigarettes per day and gets more than 100,000 nicotine "hits" per year.

Source: Russell MA, Feyerbend C. Cigarette Smoking: A Dependence on High-Nicotine Boli, Drug Metabolism Review 1978; 8(1):29-57.

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Health Care for Alaska - Continuing the Commitment

This year, at Providence Hospital, we're taking the time to celebrate and reminisce about the long history the Sisters of Providence share with Alaska . . . a rich history of risk taking and commitment that spans 85 years, from Nome to Fairbanks and Anchorage. The commitment of the Sisters to Alaska and her people is evident in the technology and facilities available at Providence and their high standard of compassion and caring for all Alaskans. On the 26th of June the Sisters of Providence dedicated the Providence Campus — the culmination of seven years of planning and construction. The works of the Sisters could never have reached this point without an effective partnership with the physicians and other health care providers in the State.

While it is appropriate to take time to celebrate the past, we must turn our focus to the future. Providence, the physicians and other health care providers throughout the state, will have many challenges to meet if they are to continue bringing health services to all those in need. Major changes are occurring in Alaskan medical care. Uncertainty about the effect of these has made all of us nervous. Not all of these changes are to our liking, but we cannot continue as we have before. The challenge will be to manage these changes so they result in better health care, either by improving quality or access, or reducing costs for patients and caregivers alike. The opportunities for success are great because of the "can do" attitude, the spirit and vision of Alaskans.

- The economy of Alaska has undergone a dramatic change over the past 18 months. Just as the supply of both professional services and inpatient resources has increased, the number of Alaskans seeking health care services has declined, leaving surplus capacity in many areas. In addition to an out-migration of people, increasing numbers of those remaining in Alaska cannot afford, and as a result are deferring, their care. This is beginning to threaten the financial position of many providers.

- The economic decline has led to drastic reductions in the state's financial support of people who need health care but are unable to pay. Most local and private community hospitals, physicians, and other local organizations treat those in need without regard for their ability to pay. If this service is to continue to be available in a declining economy, it will be imperative that decision makers, business leaders, and providers join forces to find new, innovative ways to fund categorical assistance programs. The current financial precariousness of many patients and health care providers alike dictates that a social policy must be implemented which continues support to Alaskans in need through adequately funded health entitlement programs.

- The delivery of health care itself has undergone a radical change. Many more services are being provided on an outpatient basis because of less invasive technology, new and alternative settings, and dramatic cost control pressures from government and insurers alike. In Anchorage alone, the community-wide hospital admission rate has steadily decreased from 102 admissions per thousand residents in 1980 to 79 per thousand in 1985! There will be less inpatient work in the future for physicians or hospitals and major changes in traditional settings for health care delivery.

- Business and government have targeted health care services for major cost reduction, while the demands for expensive new technology and specialized staff escalate. More complicated methods of monitoring utilization and resource consumption also contribute to rising costs. Both physicians and hospitals need increased staff to manage paperwork and obtain preauthorizations; at the same time payments are being delayed and charges challenged.

- Insurers and businesses are beginning to influence where patients go for care by restricting payments. While at the moment this seems to have increased competition in the Anchorage area only, it may in the future direct patients from the bush to Anchorage or Seattle, or to other centers Outside which would profoundly impact Alaskan health care.

- The increase in the incidence of heretofore unknown diseases, specifically immune system disorders such as AIDS, will place unprecedented pressure on all providers to serve patients well, manage costs, and provide a safe environment for employees.

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A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol — which shows how well-tolerated once-daily INDERAL LA can be.

ole therapy or concomitant therapy? ifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were treated merely with the addition of a diuretic to INDERAL LA.

or the noncompliant patients in your practice, INDERAL LA may ell be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

Control, comfort, and compliance

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

The one you know best keeps looking better

NOW AVAILABLE...

NEW LOW DOSE

INDERAL LA 60 mg

(PROPRANOLOL HCl) LONG ACTING CAPSULES

Please see next page for brief summary of prescribing information

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL the chronotropic inotropic and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock 2) sinus bradycardia and greater than first-degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

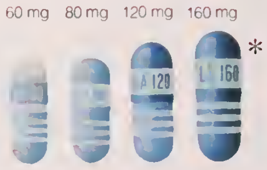
WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES



IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker In one case, this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. GENERAL Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may lead return of increased intraocular pressure

CLINICAL LABORATORY TESTS Elevated blood urea levels in patients with severe h disease, elevated serum transaminase alkaline phosphatase, lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as re pine should be closely observed if INDERAL is administered The added catecholam blocking action may produce an excessive reduction of resting sympathetic nervous act which may result in hypotension, marked bradycardia vertigo, syncopal attacks or orthost hypotension

Caution should be exercised when patients receiving a beta blocker are administere calcium-channel-blocking drug especially intravenous verapamil for both agents may press myocardial contractility or atrioventricular conduction On rare occasions, the conc tant intravenous use of a beta blocker and verapamil has resulted in serious adverse react especially in patients with severe cardiomyopathy congestive heart failure or recent myo dial infarction

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol

Ethanol slows the rate of absorption of propranolol

Phenytoin, *phenobarbitone*, and *rifampin* accelerate propranolol clearance

Chlorpromazine, when used concomitantly with propranolol, results in increased pla levels of both drugs

Antipyrine and *lidocaine* have reduced clearance when used concomitantly propranolol

Thyroxine may result in a lower than expected T_3 concentration when used concomit with propranolol

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination increasing blood levels

Theophylline clearance is reduced when used concomitantly with propranolol

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY Long-term studi animals have been conducted to evaluate toxic effects and carcinogenic potential In month studies in both rats and mice employing doses up to 150 mg/kg/day there wa evidence of significant drug-induced toxicity There were no drug-related tumorigenic ef at any of the dosage levels Reproductive studies in animals did not show any impairme fertility that was attributable to the drug

PREGNANCY Pregnancy Category C INDERAL has been shown to be embryoto animal studies at doses about 10 times greater than the maximum recommended human

There are no adequate and well-controlled studies in pregnant women INDERAL shou used during pregnancy only if the potential benefit justifies the potential risk to the fetus

NURSING MOTHERS INDERAL is excreted in human milk Caution should be exer when INDERAL (propranolol HCl) is administered to a nursing woman

PEDIATRIC USE Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have required the withdrawal of therapy

Cardiovascular Bradycardia, congestive heart failure intensification of AV block hyp sion paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually Raynaud type

Central Nervous System Light-headedness mental depression manifested by inso lassitude weakness, fatigue reversible mental depression progressing to catatonia, disturbances, hallucinations, vivid dreams, an acute reversible syndrome characterize disorientation for time and place, short- memory loss, emotional lability, sli clouded sensorium, and decreased pe mance on neuropsychometrics For imme formulations fatigue, lethargy and dreams appear dose related

Gastrointestinal Nausea, vomiting ep tric distress, abdominal cramping, diar constipation, mesenteric arterial throm ischemic colitis

Allergic Pharyngitis and agranulocy erythematous rash, fever combined with ing and sore throat, laryngospasm and res tory distress

Respiratory Bronchospasm

Hematologic Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic pur

Auto-Immune In extremely rare instances, systemic lupus erythematosus has reported

Miscellaneous Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impot and Peyronie's disease have been reported rarely Oculomucocutaneous reactions inv the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) ha been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochlorid sustained-release capsule for administration once daily If patients are switched from IND Tablets to INDERAL LA Capsules, care should be taken to assure that the desired thera effect is maintained INDERAL LA should not be considered a simple mg-for-mg substit INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitratio be necessary, especially to maintain effectiveness at the end of the 24-hour dosing inte

HYPERTENSION—*Dosage must be individualized* The usual initial dosage is INDERAL LA once daily, whether used alone or added to a diuretic The dosage m increased to 120 mg once daily or higher until adequate blood-pressure control is ach The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage mg may be required The time needed for full hypertensive response to a given dos variable and may range from a few days to several weeks

ANGINA PECTORIS—*Dosage must be individualized* Starting with 80 mg INDER once daily, dosage should be gradually increased at three- to seven-day intervals until o response is obtained Although individual patients may respond at any dosage lev average optimal dosage appears to be 160 mg once daily In angina pectoris, the valu safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few week WARNINGS)

MIGRAINE—*Dosage must be individualized* The initial oral dose is 80 mg INDER once daily The usual effective dose range is 160-240 mg once daily The dosage m increased gradually to achieve optimal migraine prophylaxis If a satisfactory response obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy be discontinued It may be advisable to withdraw the drug gradually over a period of s weeks

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are limited to permit adequate directions for use

*The appearance of these capsules is a registered trademark of Ayerst Laboratories

REFERENCES:

1. INDERAL LA National Compliance Evaluation Program. Data on file, Ayerst Laborat
2. Ravid M, Lang R, Jutrin I The relative antihypertensive potency of propranolol, oxpre atenolol, and metoprolol given once daily *Arch Intern Med* 1985, 145 1321-1323

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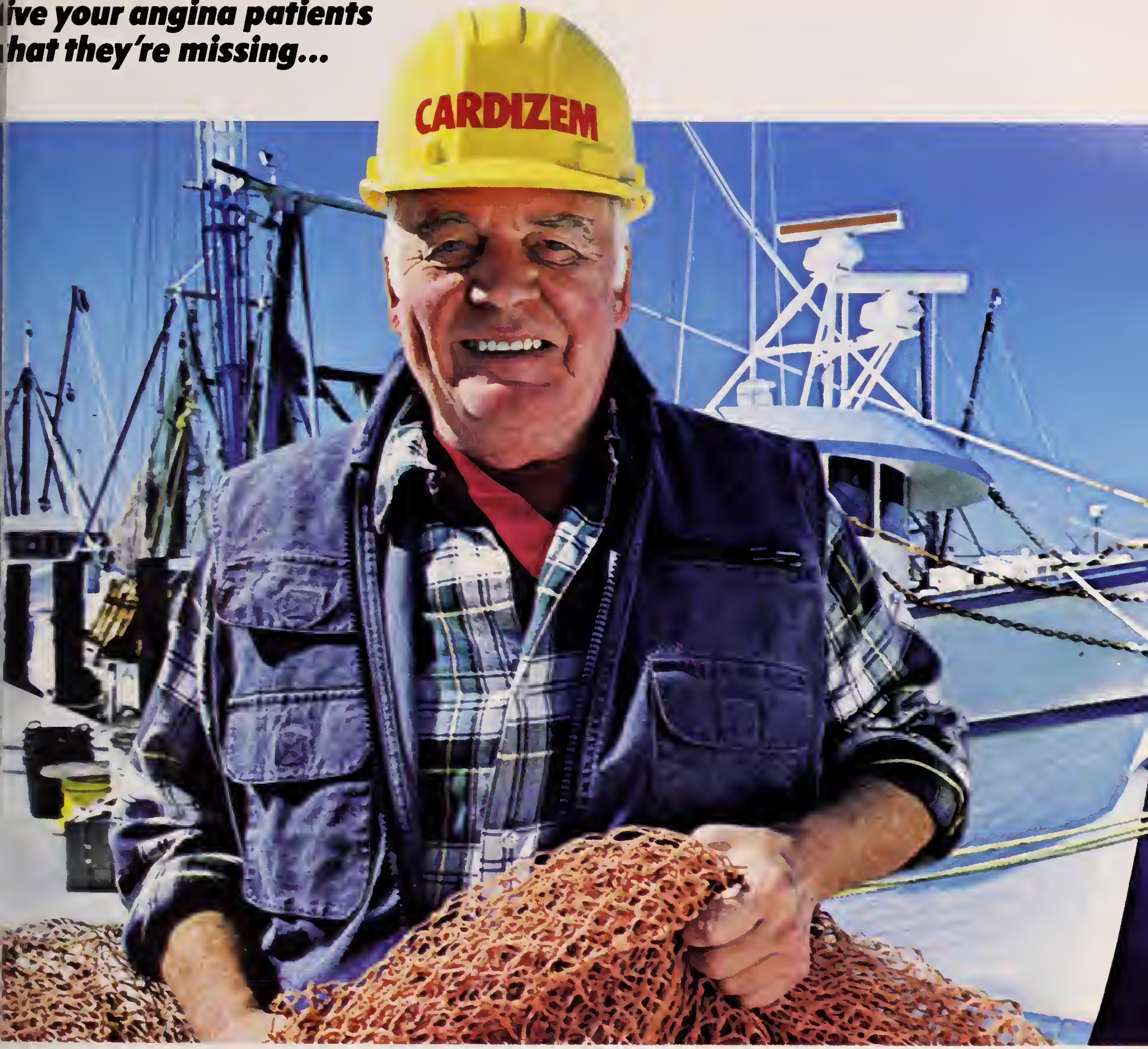

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- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

****See Warnings and Precautions.***

Please see brief summary of prescribing information on the next page.

CARDIZEM® FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

diltiazem HCl/Marion

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl) 30 mg and 60 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope
Nervous System:	Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor
Gastrointestinal:	Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase
Dermatologic:	Petechiae, pruritus, photosensitivity, urticaria
Other:	Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

Issued 7/86

See complete Professional Use Information before prescribing.

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History of Medicine in Alaska

GARY RUSSELL HEDGES, M.D., F.A.C.S.

Gary Russell Hedges was born in Ketchikan but at age 7 moved to Juneau. At the time the gold mine was still running, and he has memories of the noises—whistles, toots, explosions—as well as the minors with their lights.

Juneau was a small town of six thousand people where everyone knew everyone. Gary's father was a school teacher and coach. Gary delivered newspapers to the "ladies of the line." He remembers the territorial governors: Gruening, Heinzleman and Stepovich. At one time Waino Henderson, later to be lieutenant governor, was mayor of Juneau.

During his campaign for the presidency, Adlai Stevenson came to Juneau bringing his son who has been a candidate himself in Illinois. While father campaigned, the son fished.

Gary Hedges was graduated from Juneau High School. He had three years of math and was a good student who liked science. He remembers having his appendix out with a spinal anesthesia. He had great fun watching the operation, and having enjoyed it so much as a patient, decided that he should reverse the roles.

He is a graduate of Johns Hopkins University and went to the University of Washington to medical school. He had a rotating internship at Cleveland Metropolitan Hospital which was affiliated with Western Reserve. Then he had six years of residency in surgery at Cincinnati General Hospital.

During medical school, he served as an extern in Juneau. This was an important watershed for him since while assisting or anesthetizing patients, he decided to become a surgeon. He claims he met Margaret McCardle over an episiotomy.

Marge was born on Prince Edward Island. She studied nursing at St. Anne's Hospital. This is a Canadian order. However, the sisters were also at Fairbanks, Ketchikan and Juneau. For this reason, Marge came to Juneau.

The Hedges were married on June 7, 1961. They celebrated their twenty-fifth wedding anniversary at the Alaska State Medical Association meeting at McKinley Chalets in 1986. Gary is a former president of Alaska State Medical Association.

His great nonmedical event was winning the Salmon Derby in 1971, catching a 59 1/2 lb. fish.

Medically, he finds it most satisfying to be in a group, be it business or social, and see the successful results he has achieved. He says that it is better than being sensational at something to be surrounded by his products.

Gary enjoys the aviation industry. He has watched its development in Alaska, remembering the Pan Am flying boats in Ketchikan, working for airlines at one time, and having a memorable occasion when the atomic scientists flew into Juneau. He muses that this seems to take care of a piece of history.

He recalls Dr. C.C. Carter who had a general practice in and around Juneau. Dr. Carter owned a boat named the Three C's in which he went fishing and another Public Health Service boat in which he went from village to village "cutting." He was very good — as good as any Gary met in his training even though Dr. Carter knew only one way to perform a procedure rather than three. As with so many doctors in the earlier days of Alaska, he was called on to do more in order to save lives.

Gary mentioned Dr. Rude who, in his 90's, maintains some practice with old patients. Dr. Rude will still make an occasional housecall.

He notes among his patients Trevor Davis whose mother was an art student in England. She did anatomical drawings. Gary is proud to own two of these which were painted in 1879. Trevor Davis served on the committee which selected Alaska's flag. Of the seven members, at 93, he is the only survivor. His wife, Carol Berry Davis, now 96, played the organ in the movie house during the silent era. She still writes books, collects Tlingit songs, and has been Alaska's poet laureate. She is the author of the proposed second stanza to the Alaska Flag Song.

Gary is a Fellow of the American College of Surgeons and a member of the Pan Pacific Surgical Association which comprises Japan, Australia, Hawaii and the pacific coast of North America. He received the A.H. Robins Award for Community Service in 1986. This was well earned since he has been involved in the American Cancer Society, Juneau Receiving Home, United Way, the Territorial Sportsman Board, Boy Scouts, and Chamber of Commerce.

As a member of Pioneer Igloo #6, he is something few physicians in Alaska can be. The Pioneers of Alaska is an organization of male Alaskans who have lived in the territory/state for thirty years. Women belong to an Auxiliary which does the work. Individuals, such as Gary, born in Alaska can not join until their thirtieth birthday. It will be another two years before potential members who came after statehood are eligible.

Gwynneth Gminder Wilson
Chairman, ASMA Auxiliary History Project

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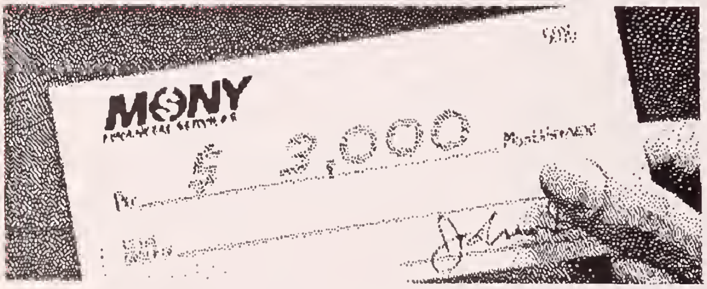

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News from the American Society for Circumpolar Health

by Robert Fortuine, M.D.[1]

ASCH Members Contribute Papers to 7th ICCH

During the 7th International Congress on Circumpolar Health, held in Umea, Sweden, from June 8–12, 1987, some 344 papers were presented on subjects related to health in the arctic and antarctic regions. Scientists and health workers from 20 nations joined together to make the week memorable for all those who participated.

Of the papers presented, 66 (19.2%) had at least one author from the United States. Among the 52 which originated from Alaska, the principal sponsors were the Arctic Investigations Laboratory of the Centers for Disease Control (13), the Alaska Department of Health and Social Services (12), the Alaska Area Native Health Service (11), Native health corporations (11), and the University of Alaska (6).

Cutting across all these agencies, institutions, and individuals were the 129 members of the American Society for Circumpolar Health, of whom 27 either authored or co-authored one or more papers. The principal authors of 39 papers were members of the society, as were one or more collaborators in 13 additional papers. In sum, 79% of the U.S. papers presented at the conference were associated with members of the society. This report will discuss in brief outline the contributions of the members of the American Society for Circumpolar Health to the 7th International Congress.

C. Earl Albrecht, the president of the First International Symposium on Circumpolar Health, chaired a panel on the future of arctic health research, in which he called for a research strategy to place greater emphasis on facilitating human adjustment in circumpolar regions and thereby improve health conditions. Ted Mala of the

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[1] Robert Fortuine, M.D., is Vice-President and Scientific Director of the American Society for Circumpolar Health.

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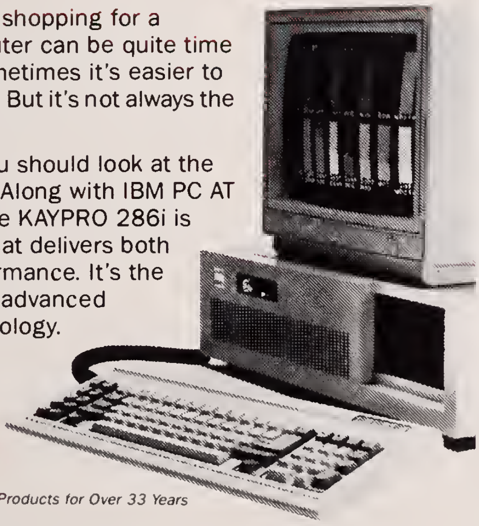

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University of Alaska (UA) presented a summary of the recent Alaskan-Siberian agreement for research collaboration in areas of common interest. S.O.E. Ebbesson (UA) gave a status report on the WAMI Program, with special attention to the type of practice ultimately chosen by its participants.

The scientific area holding the greatest interest for society members was that of infectious diseases. J.T. Wyckoff of the Yukon-Kuskokwim Health Corporation (YKHC) presented two papers on streptococcal pharyngitis, one on the use in village clinics of a special rapid detection test for group A streptococci, and the other on the results of continued streptococcal surveillance among infants and preschool children in the Bethel area. Alan Parkinson *et al.* of the Centers for Disease Control (CDC) spoke on the emerging problem of partial penicillin-resistant *Streptococcus pneumoniae* disease, based on blood and CSF isolates from the Bethel area and from Anchorage hospitals. Michael Davidson *et al.* (CDC) presented data on the unusually high prevalence of community-acquired invasive pneumococcal disease among Native infants and young children in southwestern Alaska. Ken Moss reported on four cases of *Yersinia enterocolitica* infection encountered in his Juneau pediatric practice, emphasizing the clinical variability of disease manifestations in children. Robert Wainwright *et al.* (CDC) then reviewed the total Alaskan experience since 1947 with 59 food-borne outbreaks of botulism, in which 323 cases were reported. Finally, in this section, Ann Lanier *et al.* (CDC) described the use of a new EM_2 ELISA test for the diagnosis and follow-up of alveolar hydatid disease.

All papers on viral diseases were on the subject of hepatitis B. At a workshop chaired by Brian McMahon (AANHS), Elizabeth Tower and John Middaugh of the Alaska Department of Health and Social Services (ADHSS) described the results of a large surveillance program for hepatitis B, which included not only 50,000 Alaska Natives, but also 30,000 Alaskans of other ethnic groups. Wainwright *et al.* reported on the long-term immunogenicity of hepatitis B vaccine administered to a cohort of 1630 Yupik Eskimos in 1981. McMahon *et al.* then gave a broad picture of the program for the primary and secondary prevention of hepatitis B and primary hepatocellular carcinoma in Alaska Natives. This comprehensive effort beginning in 1983 is the first of its kind to attempt to halt the spread of hepatitis B and its sequelae in a defined population. In a poster presentation McMahon *et al.* examined the effect of hepatitis B vaccine on patients testing positive only for anti-HBc and found that most such persons will have a primary rather than a booster response to the vaccine. In another poster Tower *et al.* reported on the geographical distribution of HBV serotypes in Alaska, with the finding that type ad was prevalent in the more northern parts and ay in the south. Only the former seemed to be associated with hepatocellular carcinoma. In a third poster Clare Helminiak *et al.* (AANHS) found that infants born of HBsAg positive mothers responded less well to hepatitis B vaccine than infants of seronegative mothers.

John Middaugh chaired a session called "Emergency, communication, injury, and injury prevention," at which society members contributed four papers. Stacie Pascal of the NANA Development Corporation discussed a management by objectives safety program designed for the many commercial activities carried out by NANA subsidiaries. Mark Johnson (ADHSS) presented a paper which described a long-range emergency health and communications plan, about half of which has now been implemented. In a related poster presentation he outlined the development and approval of recommendations for aeromedical services in Alaska, based on a state-wide survey of existing services and needs. M.F. Hoyle of the North Slope Borough Health Department reported on the practical application of "telehealth technology" to improve health care delivery and health-related education by linking villages with the central hospital at Barrow. In a poster he and a colleague described in greater detail the use of slow-scan video imaging to assist health aides to report illness more accurately. Middaugh presented a paper on 105 bear attacks on humans documented in Alaska since 1900, noting that campers and hikers were now supplanting hunters as victims. In a poster he reported on 94 pedestrian fatalities and 1293 injuries in Alaska from 1980-1984. Of special interest was the finding that the fatality rate among Natives was four times higher state-wide and 16 times higher in Anchorage than the rate for all others.

In the area of maternal and child health, Joan Pelto (ADHSS), using a computer program for the study of matched infant birth and death certificate information for the years 1983-85, was able to make available detailed comparisons of data across geographic and ethnic lines. Lisa Knutson *et al.* (CDC) presented a poster showing a cohort analysis of Eskimo children which demonstrated a relationship between hemoglobin levels and the number of infections in the preceding six months. A related poster by the same group compared the hemoglobin level of children in 27 villages before and after measles and pertussis epidemics swept through the area in the early 1960s. Neil Murphy *et al.* (AANHS) presented in a poster the preliminary results of screening for gestational diabetes at the Bethel Hospital, noting that 28.4% of 130 glucose screening tests showed levels above 140 mg/dl.

In the area of nutrition, Pelto and Spence, in a survey of infant feeding habits of 1066 Alaskan infants, showed that 84% of mothers responding had breast-fed their infants at some time. Among Yupik Eskimos born in the early 1960s, Knutson *et al.* found that 60% had received supplemental foods by the age of four months, and 80% had received meat, fish, or soup by the time they were eight months of age. In adult nutrition, A.L. Cruz *et al.* (CDC) presented a poster which described the results of a dietary survey in a coastal and a riverine Eskimo village to show the intake of marine fish, seal oil, and salmon, as correlated with blood lipid patterns. The same villages were the subject of another poster by Hall *et al.*, who examined in greater detail the dietary preferences of the Eskimos during the late summer months.

Arctic medical history and traditional medicine was the topic of a session chaired by Robert Fortuine (AANHS). Murphy *et al.* reviewed a selection of Yupik traditional healing methods and attempted to explain their rationale. Fortuine presented a survey of empirical healing methods of the Alaska Natives, based on historical sources. In a second paper he traced the influence of scurvy on the early exploration and settlement of Alaska.

In the area of health care planning and delivery, Middaugh presented mortality data expressed in terms of years of potential life lost (YPLL) and demonstrated that involuntary injury led all other causes by a large margin. Robert Burgess *et al.* (AANHS) demonstrated in a poster session the 1987 edition of the *Community Health Aide/Practitioner Manual,* which has been under development for several years. Murphy and Schraer reported on their experience field-testing under winter conditions a portable reflectance meter glucose monitoring device.

Cheryl Mann (UA) presented a paper on the dynamics of being raised in an alcoholic family system, particularly the impact on children as they grow to adulthood. Norma Forbes and V. van der Hyde (ADHSS) reported on the unusually high suicide rate in Alaska, especially among young adult Alaska Natives, who have a rate nearly eight times the national average. W.G. Hlady and Middaugh made a careful study of suicide deaths for 1983–84 based on death certificates and validated by coroners' files and autopsy records. They were able to demonstrate that the use of firearms, associated with alcohol intoxication, was the principal means in all groups studied.

In the field of chronic diseases, society members presented or collaborated on several papers. G.S. Boyer *et al.* (AANHS) reviewed known cases of rheumatic diseases among Alaskan Eskimos and Indians and found a high incidence of rheumatoid arthritis among Tlingit women, as well as unusual patterns of Reiter's syndrome, incomplete Reiter's, and ankylosing spondylitis among various Native groups. Davidson *et al.* compared the incidence of influenza/pneumonia, tuberculosis, lung and laryngeal cancer, and chronic lung disease among the various subgroups of the Alaskan population. A.B. Way of the Texas Tech Health Sciences Center collaborated on two papers with several Icelandic scientists to study the incidence of cardiovascular disease and the pattern of blood lipids among individuals of Icelandic descent living in Manitoba.

In a session chaired by Ann Lanier, Susan Clift *et al.* (AANHS) reported on five cases of anaplastic carcinoma of the salivary glands which were positive for EBV by serology, immunofluorescent staining, or by DNA hybridization. McMahon *et al.* presented the results of periodic screening of HBV carriers by alpha-fetoprotein determinations, and found 10 cases of hepatocellular carcinoma identified by this method over a four-year period. In another session Lanier *et al.* described current findings from a state-wide cancer surveillance system for Alaska Natives, with particular emphasis on the differing cancer incidence patterns by site between Natives and other population groups.

Bruno Kappes (UA) presented two papers on frostbite and cold physiology. In the first, he and his colleague described the use of infrared measurements of cross-country skiers' hand temperatures and attempted to relate their findings to performance in the Iditaski race. In a second paper he and W.J. Mills discussed the use of the Minnesota Multiphasic Personality Inventory to describe psychological patterns in 30 cases of frostbite. R.G. Foutch (U.S. Army) and Mills presented a review of traditional Native methods for dealing with frostbite, hypothermia, and snow blindness.

Middaugh *et al.* compared the incidence of otitis media among various racial groups in Alaska and found a marked improvement in the problem among Natives in recent years. All Alaskans had a high incidence of high-frequency hearing loss, particularly Eskimo and Indian men.

Raymond Dieter *et al.* (Glen Ellyn Clinic, IL) studied the patterns of ocular disease among Alaska Native teenagers at the Mount Edgecumbe boarding school in the years 1961–62, as compared with 1953–54. In the later period the chief problems were refractive errors, corneal scarring, blepharitis, corneal transplants, and conjunctivitis.

This rapid survey of some of the research findings presented at the 7th ICCH shows the diversity of topics which are under investigation by Alaskan researchers. Much more remains to be done, however, particularly more of the excellent collaborative work among federal and state agencies, Native organizations, and private individuals.

The full text of many of the studies described above will be published in a special issue of *Arctic Medical Research* in approximately one year.

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RESOLUTIONS
Adopted by House of Delegates
Alaska State Medical Association
Annual Meeting, June 3–7, 1987

Resolution No. 87–1
SUBJECT: Supporting Physician Prescribing and Pharmacist Dispensing

WHEREAS, the Alaska Pharmaceutical Association has requested that the Alaska State Medical Association take a position on physician dispensing; and

WHEREAS, this subject is under nationwide publicity; and

WHEREAS, federal legislation is pending on this subject; and

WHEREAS, the American Medical Association has suggested a rational approach; therefore

BE IT RESOLVED that generally physicians prescribe and pharmacists dispense, with allowances for special situations of geography, time and patient convenience.

Resolution No. 87–2
SUBJECT: Worker's Compensation Insurance

WHEREAS, Alaska's Worker's Compensation insurance premiums are the highest in the nation; and

WHEREAS, an employer coalition has formed to consider legislative action to decrease these costs; and

WHEREAS, the coalition is requesting advice from the Alaska State Medical Association on how to improve medical aspects of worker's compensation after a worker is injured; therefore

BE IT RESOLVED that the Alaska State Medical Association explore methods of assisting employers, insurers, and the Compensation Board in assessing quality and appropriateness of care rendered to injured workers in Alaska.

Resolution 87–3
SUBJECT: Updating Alaska State Medical Association Policy Statements

WHEREAS medical, social, economical, political, and cultural issues have developed dramatically over the past two decades; and

WHEREAS, some of the minutes of early Alaska State Medical Association annual meetings leave votes on resolutions unclear or unknown; therefore

BE IT RESOLVED that the Alaska State Medical Association recognizes resolutions passed prior to 1968 for continued historical interest, and now orders that none of those resolutions are current Alaska State Medical Association policy, with the specific exceptions of Resolution 60–24, Conferring Honorary Membership; 61–11, Directing Hiring of a Legislative Representative; 62–3, Endorsing an Alaska State Medical Political Action Committee; and 62–18, Directing that the Legislative Representative be physically in the capital city during the Legislative Session.

Resolution 87–4
SUBJECT: Supporting Alaska State Medical Association Membership for Physicians Only

WHEREAS, the Health Association of Alaska (nee Alaska Hospital Association) has had an invited, non-voting representative at many Alaska State Medical Association council meetings; and

WHEREAS, both voting at the Alaska State Medical Association annual meeting and service on the Alaska State Medical Association council is presently restricted to active member physicians only; and

WHEREAS, there has been discussion and previous action on Resolution 84–31 asking for drafting a bylaws change that would permit a non-physician to be a member of the council and to vote at the Alaska State Medical Association annual meeting; therefore

BE IT RESOLVED that the Alaska State Medical Association with this action revokes Resolution 84–31 and reaffirms that both membership on the council and voting at the Annual Meeting are reserved for active physician members of the Alaska State Medical Association.

Resolution 87–6
SUBJECT: Combining Alaska State Medical Asso-

ciation and Alaska Academy of Family Physicians Conventions

WHEREAS, the Alaska Chapter, Academy of Family Physicians and the Alaska State Medical Association are holding separate conventions and this detracts from both organizations; therefore

BE IT RESOLVED that an ad hoc committee be appointed to arrange combined convention and scientific congresses in the future starting in 1988 or 1989.

Resolution 87–7
SUBJECT: Thanking the Speakers

WHEREAS, those attending the Alaska State Medical Association annual meeting in June of 1987 were educated, informed and entertained by a series of excellent speakers; and

WHEREAS, the speakers individually took time away from their families and their day–to–day responsibilities to speak to us; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the speakers and by this resolution acknowledges their vital role in making the Alaska State Medical Association 1987 annual meeting in Juneau a success.

Resolution 87–8
SUBJECT: Thanking the Exhibitors

WHEREAS, those attending the Alaska State Medical Association annual meeting in June of 1987 were educated, informed and entertained by a series of excellent speakers; and

WHEREAS, the Alaska State Medical Association annual meeting in June 1987 was enhanced and indeed enabled by an outstanding facility with a responsive and efficient staff; and

WHEREAS, the exhibitors brought to Juneau the widest range of opportunities, ideas, services, pharmaceuticals and devices ever made available at an Alaska State Medical Association annual meeting; and

WHEREAS, neither the speakers nor the facility would have been feasible without significant financial contributions by exhibitors and contributors, therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the exhibitors and by this resolution acknowledges their vital role in making the Alaska State Medical Association 1987 annual meeting in Juneau a success.

Resolution 87–9
SUBJECT: Thanking the Contributors

WHEREAS, those attending the Alaska State Medical Association annual meeting in June of 1987 were educated, informed and entertained by a series of excellent speakers; and

WHEREAS, the Alaska State Medical Association annual meeting in June 1987 was enhanced and indeed enabled by an outstanding facility with a responsive and efficient staff; and

WHEREAS, neither the speakers nor the facility would have been feasible without significant financial contributions by exhibitors and contributors, therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the contributors and by this resolution acknowledges their vital role in making the Alaska State Medical Association 1987 annual meeting in Juneau a success.

Resolution 87–10
SUBJECT: Thanking the Juneau Convention Center

WHEREAS, those attending the Alaska State Medical Association annual meeting in June of 1987 were enhanced and indeed enabled by an outstanding physical facility with a responsive and efficient staff; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the Juneau Convention Center staff and by this resolution acknowledges their vital role in making the Alaska State Medical Association 1987 annual meeting in Juneau a success.

Resolution 87–11
SUBJECT: Thanking the Juneau Folk Ensemble and John d'Armond

WHEREAS, the Juneau Folk Ensemble sang and danced, in Russian costume, with great exuberance and harmony; and

WHEREAS, John d'Armond lead the singers and shared interesting slides of Russia; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the Juneau Folk Ensemble and John d'Armond and by this resolution acknowledges their role in making the Alaska State Medical Association 1987 annual meeting in Juneau so much fun.

Resolution 87–12
SUBJECT: Thanking the Thane Ore House and the Gold Nugget Revue

WHEREAS, those attending the Thane Ore House enjoyed a superb dinner and lively entertainment by the Gold Nugget Revue; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the cooks, cancan dancers, actors and singers and by this resolution acknowledges their role in making the Alaska State Medical Association 1987 annual meeting in Juneau enjoyable.

Resolution 87–13
SUBJECT: Thanking Janice and Butch Holst

WHEREAS, the meals were superb, the entertainment outstanding, the cruise was spectacular (for those hearty sailors), the President's Dinner Ball was elegant, and the coordination of all events went remarkably well; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to Janice Holst and Butch Holst and by this resolution acknowledges the many hours spent in coordinating this event and their vital role in making the Alaska State Medical Association 1987 annual meeting in Juneau a success.

Resolution 87–14
SUBJECT: Thanking Medical Insurance Exchange of California

WHEREAS, the reception before the President's Dinner Ball was well attended and much laughter and conversation was heard reminiscing week's events; and

WHEREAS, Medical Insurance Exchange of California was so generous and gracious to sponsor the President's Reception; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the Medical Insurance Exchange of California and by this resolution acknowledges their role in making the Alaska State Medical Association 1987 annual meeting in Juneau enjoyable.

Resolution 87–15
SUBJECT: Syntex Laboratories

WHEREAS, Syntex was not only a participant and exhibitor; but also sponsored the delicious soup and salad bar luncheon; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to Syntex for their generosity, and by this resolution acknowledges their role in making the Alaska State Medical Association 1987 annual meeting in Juneau a success.

Resolution 87–16
SUBJECT: Thanking Bob Garrison – Guide

WHEREAS, Bob Garrison gave of his time to guide the many intrepid hikers up the trail to Perseverance Mine; and

WHEREAS, he explained about flora and fauna of the area; and

WHEREAS, he increased the historical knowledge of the participants; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks and by this resolution acknowledges his role in making the Alaska State Medical Association 1987 annual meeting in Juneau a healthful experience.

Resolution 87–17
SUBJECT: Thanking Glacier Express

WHEREAS, Glacier Express extended to convention participants the "ride of a lifetime" to the twin Sawyer Glaciers in Tracy Fjord; and

WHEREAS, several cruise "seapersons" spent most of their time looking over the stern of the boat "feeding the fish"; and

WHEREAS, the scenery was magnificent, the seals carefree, the water a calm and shimmering sea; therefore

BE IT RESOLVED that the Alaska State Medical Association expresses its sincere thanks to the crew of the Glacier Express and by this resolution acknowledges their role in making the Alaska State Medical Association 1987 annual meeting in Juneau an unforgettable experience.

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RACE RESULTS
4TH ANNUAL AAFP FUN RUN

JUNEAU, ALASKA JUNE 6, 1987

1.	Bruce Palmer	25:01
2.	Douglas G. Smith	25:03
3.	Len Ceder	25:04
4.	Aaltje Smith	26:22
5.	James Dunn	26:33
6.	Sue Scott	26:35
7.	Ron Tinsley	27:08
8.	Jerry Little	29:25
9.	Chris Wegener	29:43
10.	Ron Christensen	29:46
11.	Vivian Pederson	30:43
12.	Joan James	31:33
13.	Peter McKeown	35:33
14.	Lorrie Horning	38:36
15.	Betty Elsner	38:53
16.	Terri Patterson	39:33
17.	Carol Craig	40:15
18.	Diane Waterworth	50:12
19.	Frances Angelo	53:59
	Judy Schalow	53:59

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State Medical Board

In the closing days of this year's legislative session HB70, which significantly alters the functioning and expands the authority of the State Medical Board, was passed unanimously by the House and Senate. The measure, which was introduced by Representative John Sund of Ketchikan after consultation with the State Board and ASMA, passed with the strong support of both groups and with input from the hospital association. With appropriate modification to meet local needs and the structure of law and precedent in the State of Alaska, it follows recommendations for model legislation developed at the national level by the Federation of State Medical Boards. It is also in conformity with federal mandates that will go into effect in 1989 under the Health Care Quality Improvement Act of 1986 (PL-99-660).

Pivotal in the structuring and eventual passage of the law was the innovative and dedicated work of the Division of Occupational Licensing and its enthusiastic Director, Ms. Kathy Marshall. The input of ASMA via its Legislative Committee under David McGuire, M.D. and many others including President, Don Thieman, M.D.; Doug Smith, M.D.; David Johnson, M.D.; Executive Director, Ray Schalow and lobbyist, Rick Urion was invaluable in convincing the legislature that this was a good faith, committed effort at effective self-regulation. Everyone involved can be proud of the final product.

Key provisions of the legislation are as follows:

1. *Fees and Services are Placed in Balance:* The legislation mandates that the fees charged for licensure will reflect actual costs to the state for administering and regulating the practice of medicine. As a corollary, the obverse, that the fees charged will reflect the services rendered is also mandated. The language was carefully crafted to ensure that the Medical Board (and other boards, for this section of the bill affects all boards) could be run on zero-based budgeting and effectively respond to its needs by regulating fees without offending the prohibition of dedicated funding called for by Article IX of the Alaska Constitution.

2. *The authorization of the State Medical Board is Extended to June 30, 1991* — The board was due to sunset June 30, 1987, and this provision extends its life in conformity with law.

3. *The Board will Coordinate with Private Professional Organizations to Establish an Impaired Professionals Program.* This gives the board the authority to set up programs, most likely in association with ASMA, to offer help to physicians impaired by drugs or alcohol while protecting the public by ensuring that the full weight of the law will descend on those refusing to cooperate or failing to comply with a rehabilitation program mandated to preserve licensure. The program envisioned will be a monitored treatment program. It is hoped its existence will eventually induce those in trouble to seek help before they come to the attention of the board.

4. *The Board will be Assigned the Services of a Dedicated Investigator and an Executive Secretary.* This provision, in conjunction with the provision matching fees to service and service to fees, will form the cornerstone on which improved board functioning will rest. Having day-to-day services of these key personnel should tie together administration, investigations and enforcement, and direction of the impaired physician program and bring the regulation of medicine in Alaska into the 20th century. A volunteer board is great for setting policy, but it requires dedicated personnel to supervise day-to-day operations.

5. *License Renewal Shall be Biannual.* This replaces the present quadrennial system which is unwieldy and provides inadequate data for the setting of appropriate fees to fund the function.

6. *The Board Shall Report Disciplinary Actions and Refusals to Grant or Renew Licenses to the Federation of State Medical Boards Disciplinary Data Bank.* This is in conformity with PL-99-660 and ensures that final actions taken in Alaska will be reported nationally. Such permits us to advise other states of our disciplinary cases and to receive such information in return. The idea in this is to participate in the national effort to thwart those who try to jump from jurisdiction to jurisdiction ahead of the law and the actions of the various licensing boards.

7. *Physicians and Hospitals Required to Report:* Physicians who treat other physicians for alcohol or drug addiction or mental, emotional or personality disorders will be required to report this to the board if there is probable cause to believe that the treated individual constitutes a danger to his or her patients

or the public if he or she continues in practice. Again the wording is carefully crafted to permit confidential treatment without board notification of such individuals if they are compliant with the treatment program devised. It is envisioned that the treatment program set up between the board and the private professional organizations (read ASMA) will be a monitored one and accept self-referrals and referrals from ASMA's Impaired Physicians Committee on an "anonymous" basis and mandated referrals from the board when such impairment comes to board attention. It would be anticipated that those entering the program "anonymously" would only be brought to board attention for noncompliance with or failure on monitored treatment.

8. *Hospitals Required to Report Unfavorable Actions.* A hospital that revokes, suspends, conditions, restricts, or refuses to grant hospital privileges or imposes a consultation requirement on a physician licensed in Alaska is required to report this to the board within seven working days of the action. Additionally physicians resigning staff privileges while under investigation of a matter that could lead to any of the above actions must be reported. A consultation requirement is defined as a new restriction put on existing privileges so that new staff consultation requirements used by many hospitals are not to be reported nor are actions taken solely for failure to attend meetings or complete medical records.

9. *Good Faith Reporting Exempt from Civil or Criminal Liability.* A physician or hospital which submits a report in a good faith under items 7 and 8 above is exempt from criminal and civil liability.

10. *Hospitals and Physicians may not Refuse to Submit a Report or Withhold Evidence* on the grounds that

    a. The report concerns a matter disclosed in a physician-patient relationship or during a meeting of a hospital medical staff, governing body or committee; or
    b. the report is required to be kept confidential under AS 18.23.030, the statute covering the confidentiality of records of review organizations (peer-review committees).

11. *Material Received from Peer-Review Committees held Non-discoverable.* Fearing that transmission of peer-review information to the state board would put a chill on such review, AS 18.23.030 is further altered such that information gathered during a board investigation is held non-discoverable and not subject to subpoena unless and until the board takes action to suspend, revoke, limit or condition the license of the person who is the subject of the report or investigation (at which time it becomes a matter of public record in any case.)

12. *Failure of a Hospital to Comply with the Orders of the Board to Supply Information During an Investigation may Result in Action Against the Hospital's License.* The Department of Health and Social Services is empowered to deny, suspend or revoke a hospital's license for failure to comply with the requirements to report outlined above. This puts teeth into the requirement and, along with the immunity from liability for good faith reporting, serves to protect hospitals which in the course of things would wish to report but have been hesitant to do so in the past for fear of suit.

13. *A One Time Surcharge is to be Imposed on Presently held Licenses to Cover Cost of New Positions Until the Next Renewal Date — December 31, 1988.* This is to implement the new program in the interim between passage of the bill and the setting of new fees for licenses renewed after December 31, 1988. The board has determined this one time surcharge will be $125. It is anticipated that in the future biannual licensure will run about $550-600 ($275-300/year).

Questions about application of the new provisions are welcome as are suggestions to the board about utilization of the services of the new personnel and start-up of an impaired physicians program. Our number in Juneau is 465-2541 (Kurt West, Licensing Examiner, Pouch D, Juneau, Alaska, 99811) and in Anchorage the number is 561-2878 (Gary Dodson, Investigator); or you may contact any of the board members (listed below).

Thomas L. Conley, M.D.
3612 Tongass Avenue
Ketchikan, Alaska 99901
225-5146

Bonnie Coghlan
741 8th Avenue
Fairbanks, Alaska 99701
452-1165

Jeffrey A. Partnow, M.D.
1919 Lathrop, Drawer 2
Fairbanks, Alaska 99701
452-4769

Abigale Hensley
P. O. Box 710
Kotzebue, Alaska 99752
442-3669

James W. Thompson, M.D.
SEARHC Medical Clinic
3258 Hospital Drive
Juneau, Alaska 99801
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George S. Rhyneer, M.D.
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ALASKA'S RESOURCES

ALASKA'S FUTURE

A resource is something we can turn to for support or help. The most prominent resource in Alaska in the past 10 years has been oil: development of the Prudhoe Bay oil field has been responsible for unparalleled growth and change. But the collapse of oil prices in 1986 brought that growth to a shuddering halt and reminded Alaskans that we need to fit all our resources together to shape the future.

Alaska's resources range from petroleum and fish to scenic beauty and wildlife to the Permanent Fund and the state's dwindling but still significant petroleum income. Other resources are less tangible but no less important: the ingenuity and resourcefulness of Alaskans themselves. The 1987 Arctic Science Conference presents an excellent opportunity to evaluate the changes of the past decade and to begin thinking about how to combine our resources to meet the changes the next decade holds for us.

1987 ARCTIC SCIENCE CONFERENCE

(38TH ALASKA SCIENCE CONFERENCE)

American Association for the Advancement of Science – Arctic Division

American Society for Circumpolar Health
This society will hold its annual meeting during a noon luncheon, Thursday, September 24.
The cost for the lunch will be $15.00.

SEPTEMBER 24-26, 1987

SHERATON ANCHORAGE HOTEL
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My Turn

Oxymorons and Medicine

- Physicians are told by attorneys, judges, legislators, insurance companies, the media, and government to improve self-regulation of our profession. We are to clean up medicine by taking action against incompetent physicians to limit or remove them from practice.
    - Provide no indemnity so that physicians who initiate actions, although acting in good faith, may be sued for slander, libel, and antitrust. Provide the court system and attorneys access to peer-review minutes that were to have been strictly confidential. See to it that the State Medical Board is under-staffed and under-funded so that it is unable to investigate complaints and allegations brought against physicians.
- Force highly trained and skilled physicians out of the delivery room through tort litigation and astronomical malpractice premiums.
    - License lay midwives and establish state sanction for other untrained or partially trained individuals to "assist" at childbirth.
- Complain about health costs and the quality of physician care.
    - Develop a massive administrative bureaucracy at a conservative cost of one billion dollars per year that delivers not one iota of health care or service. Continually expand this bureaucracy without regard to its cost and exempt it from scrutiny, including cost-benefit and cost-effective analysis. Whenever possible, confuse "costs" with "charges."
- Require pre-hospitalization review in order to contain costs.
    - Give lip service to preserving quality and don't try to measure it. Allow chiropractors to gain hospital admission privileges.
- Ask physicians voluntarily to freeze fees and then freeze them anyway.
    - Raise malpractice premiums 73% in 1987. Pay physicians 80% of their charges and then make it illegal for physicians to bill their patients for the difference.
- Require a certificate-of-need for health facility construction.
    - Exempt from certificate-of-need requirements the federal government, including the military, veterans administration, and Indian Health Service.
- Have Congress pass the Alaska Native Claims Settlement Act (ANCSA) in 1971. Provide in Section 2(b) that "The settlement should be accomplished rapidly without establishing any permanent racially defined institutions, rights, privileges."
    - Build in 1988-90 a new Alaska Native Medical Center. Let Anchorage in the 1990's have to contend with three 120 million dollar medical centers plus a new Elmendorf AFB hospital.
- Establish health objectives for the nation, stressing health promotion and disease prevention
    - Refuse to cover counselling, patient education, periodic screening tests, etc. in health insurance plans. Provide lucrative reimbursement for invasive procedures, instrumentation, high-tech, and tests.
- Continually attack physicians as the cause of high health care costs
    - Ignore that physicians salaries account for no more than 10-12% of the nation's health dollar. Avoid examination of costs and charges for laboratory tests, drugs, and hospital supplies. Who *says* 10% of the GNP is enough for health care?
- Pledge governmental concern over quality of health services
    - Develop and encourage programs—primarily health care financing programs—that restrict individuals' ability to select their own physician.

John Middaugh, M.D.
Anchorage, AK

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President's Page

We have just completed the Forty-second Annual Convention of ASMA. As Convention Committee Chairman, I fear I may have a biased opinion, but I think we had an outstanding convention this year. From all the comments that I have heard, I feel the CME was excellent. Most of the speakers were from the University of Washington Medical School. We had two Alaskan physicians give talks, Dr. Charles Sternhagen and Dr. John Wrigley. Syntex Laboratories presented a symposium on various arthritides and brought up two speakers. This was the first time we had gone through a medical school for most of the speakers, and I certainly would recommend this measure for next year's convention.

The business sessions were handled promptly and efficiently by Dr. Don Thieman, our immediate past-president. At the council meeting on June 7, 1987, it was decided that next year's convention in Fairbanks would be held a week later so as not to coincide with the end of the school year in Anchorage. It was also decided, and I wholeheartedly agree with this plan, to have a committee work with the Alaska Academy of Family Physicians to develop a conjoint convention, either in 1988 or 1989. Along with this plan it was decided to hold the 1989 convention in Sitka. We look forward to working with the AAFP in developing such a meeting. I think it would be a good step forward for both organizations.

I would be remiss if I did not extend our thanks and appreciation to the members of the ASMA Auxiliary for the projects they have carried out in the past year. Patrice Gerster is the immediate past-president of the Auxiliary, and I must say that she did an excellent job in carrying out a project to fund the Asthma Camp for this summer. Again, our thanks to all the members of the Auxiliary.

Some of the other highlights of the meeting in Juneau consisted of the exhibitors' reception hosted by ASMA. I think that the exhibitors were certainly appreciative of this step. I think a great deal of that credit goes to Ray Schalow, our executive director.

Other highlights included the Glacier Express cruise which certainly separated the "seafarers" from the "upchuckers." The president's banquet was well attended and, all in all, was a very delightful evening. Lieutenant Governor Steve McAlpine brought us some words of wisdom. I think basically he was asking for the physicians and attorneys in this state to work toward a compromise regarding TORT reform. There was a Scottish bagpipe band for entertainment before dinner and a very well-received, 17-piece, Glenn Miller type band for post-banquet dancing.

As the newly installed president of ASMA, I look forward to a very challenging year. I certainly hope that I can continue to handle the rough spots as expediently and as competently as Don Thieman. To my mind, there are several areas in which we need to continue to work as the voice for organized medicine in the state of Alaska.

Number one, of course, is continued efforts for TORT reform. I feel that our next effort should be in the way of developing an initiative to be placed on the ballot in the fall of 1988. This should get the attention of the legislators in the next session and, hopefully, spur them to pass SB-22. If this plan is to be successful, the efforts to get this initiative on the ballot will need to be started now. We certainly look forward to working with the Citizens Coalition for Tort Reform in this area.

Another measure that was not resolved at this annual meeting was the idea of unification with the AMA. Unification, as you are aware, means that if you join the local and state medical association, you also have to join the AMA. This motion was tabled at the meeting in 1986 and is effectively still tabled, but this eventually will have to be dealt with, either at next year's meeting or the year after I would assume. To my own way of thinking, I feel that if we can increase our membership in ASMA along with an increase in the percentage of members who also belong to the AMA, we can accomplish the same objectives as unification without creating such a divisive policy.

The third goal we should strive to effect is that of increased membership. Hopefully, we will have a good strong membership committee for the coming year. One of the complaints about ASMA's annual meetings in the past has been the lack of CME, particularly for the primary care physicians. I think those complaints have been answered very well in these past two meetings. Certainly there is no reason why we can't continue to provide excellent CME sessions in the ensuing years.

Last, but certainly not least, a thank you to Ray Schalow, Gayle, Verna, and Lou Anne for their efforts in making this convention such an outstanding success. I look forward to working with you this coming year.

Jerry Little, M.D.
President
Alaska State Medical Association

e prescribing, see complete prescribing information in SK&F CO. ture or *PDR*. The following is a brief summary.

RNING

s drug is not indicated for initial therapy of edema or hyperten- n. Edema or hypertension requires therapy titrated to the individual. his combination represents the dosage so determined, its use y be more convenient in patient management. Treatment of hyper- sion and edema is not static, but must be reevaluated as con- ons in each patient warrant.

aindications: Concomitant use with other potassium-sparing agents as spironolactone or amiloride. Further use in anuria, progressive or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum sium. Hypersensitivity to either component or other sulfonamide- ed drugs.

ings: **Do not use potassium supplements, dietary or otherwise, s hypokalemia develops or dietary intake of potassium is markedly ired.** If supplementary potassium is needed, potassium tablets d not be used. Hyperkalemia can occur, and has been associated cardiac irregularities. It is more likely in the severely ill, with urine e less than one liter/day, the elderly and diabetics with suspected nfirmed renal insufficiency. Periodically, serum K^+ levels should be mined. If hyperkalemia develops, substitute a thiazide alone, restrict ntake. **Associated widened QRS complex or arrhythmia requires pt additional therapy.** Thiazides cross the placental barrier and ar in cord blood. Use in pregnancy requires weighing anticipated lits against possible hazards, including fetal or neonatal jaundice, bocytopenia, other adverse reactions seen in adults. Thiazides ar and triamterene may appear in breast milk. If their use is essential, atient should stop nursing. Adequate information on use in children t available. Sensitivity reactions may occur in patients with or with- history of allergy or bronchial asthma. Possible exacerbation or ation of systemic lupus erythematosus has been reported with ide diuretics.

autions: The bioavailability of the hydrochlorothiazide component of zide' is about 50% of the bioavailability of the single entity. retically, a patient transferred from the single entities of triamterene ydrochlorothiazide may show an increase in blood pressure or fluid tion. Similarly, it is also possible that the lesser hydrochlorothiazide ailability could lead to increased serum potassium levels. However, sive clinical experience with 'Dyazide' suggests that these conditions not been commonly observed in clinical practice. Angiotensin- erting enzyme (ACE) inhibitors can elevate serum potassium; use caution with 'Dyazide'. Do periodic serum electrolyte determinations icularly important in patients vomiting excessively or receiving nteral fluids, and during concurrent use with amphotericin B or costeroids or corticotropin [ACTH]). Periodic BUN and serum tinine determinations should be made, especially in the elderly, etics or those with suspected or confirmed renal insufficiency. ulative effects of the drug may develop in patients with impaired renal tion. Thiazides should be used with caution in patients with impaired tic function. They can precipitate coma in patients with severe liver ase. Observe regularly for possible blood dyscrasias, liver damage, idiosyncratic reactions. Blood dyscrasias have been reported in nts receiving triamterene, and leukopenia, thrombocytopenia, ulocytosis, and aplastic and hemolytic anemia have been reported thiazides. Thiazides may cause manifestation of latent diabetes tus. The effects of oral anticoagulants may be decreased when concurrently with hydrochlorothiazide; dosage adjustments may be ssary. Clinically insignificant reductions in arterial responsiveness repinephrine have been reported. Thiazides have also been shown to ase the paralyzing effect of nondepolarizing muscle relaxants such bocurarine. Triamterene is a weak folic acid antagonist. Do periodic studies in cirrhotics with splenomegaly. Antihypertensive effects be enhanced in post-sympathectomy patients. Use cautiously in cal patients. Triamterene has been found in renal stones in associa- with the other usual calculus components. Therefore, 'Dyazide' ld be used with caution in patients with histories of stone formation. w occurrences of acute renal failure have been reported in patients Dyazide' when treated with indomethacin. Therefore, caution is sed in administering nonsteroidal anti-inflammatory agents with zide'. The following may occur: transient elevated BUN or creatinine th, hyperglycemia and glycosuria (diabetic insulin requirements may tered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), easing alkali reserve with possible metabolic acidosis. 'Dyazide' feres with fluorescent measurement of quinidine. Hypokalemia is mmon with 'Dyazide', but should it develop, corrective measures ld be taken such as potassium supplementation or increased dietary e of potassium-rich foods. Corrective measures should be instituted iously and serum potassium levels determined. Discontinue correc- measures and 'Dyazide' should laboratory values reveal elevated m potassium. Chloride deficit may occur as well as dilutional natremia. Concurrent use with chlorpropamide may increase the risk evere hyponatremia. Serum PBI levels may decrease without signs hyroid disturbance. Calcium excretion is decreased by thiazides. zide' should be withdrawn before conducting tests for parathyroid tion. Thiazides may add to or potentiate the action of other anti- rtensive drugs. Diuretics reduce renal clearance of lithium and ease the risk of lithium toxicity.

erse Reactions: Muscle cramps, weakness, dizziness, headache, mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other natological conditions; nausea and vomiting, diarrhea, constipation, er gastrointestinal disturbances; postural hypotension (may be ravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, esthesias, icterus, pancreatitis, xanthopsia and respiratory distress uding pneumonitis and pulmonary edema, transient blurred vision, adenitis, and vertigo have occurred with thiazides alone. Triamterene been found in renal stones in association with other usual calculus ponents. Rare incidents of acute interstitial nephritis have been orted. Impotence has been reported in a few patients on 'Dyazide', ough a causal relationship has not been established.

plied: 'Dyazide' is supplied as a red and white capsule, in bottles of 0 capsules; Single Unit Packages (unit-dose) of 100 (intended for titutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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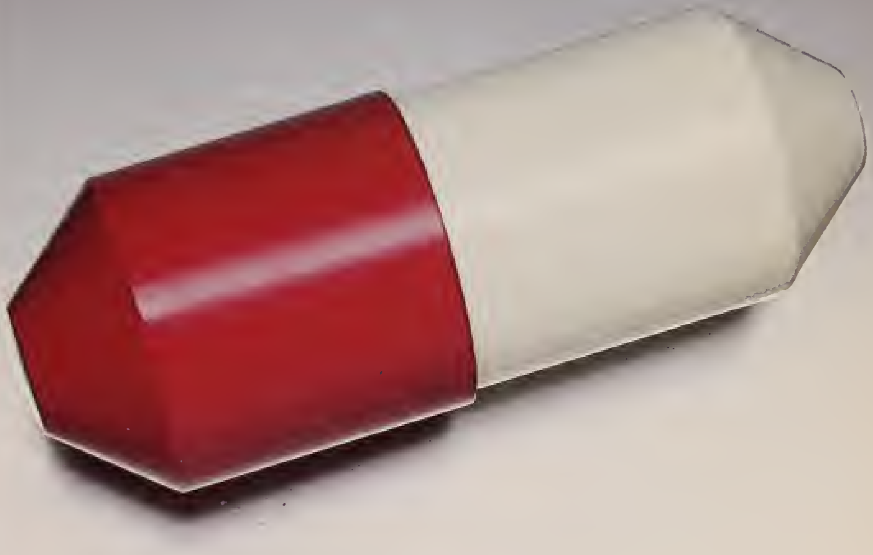

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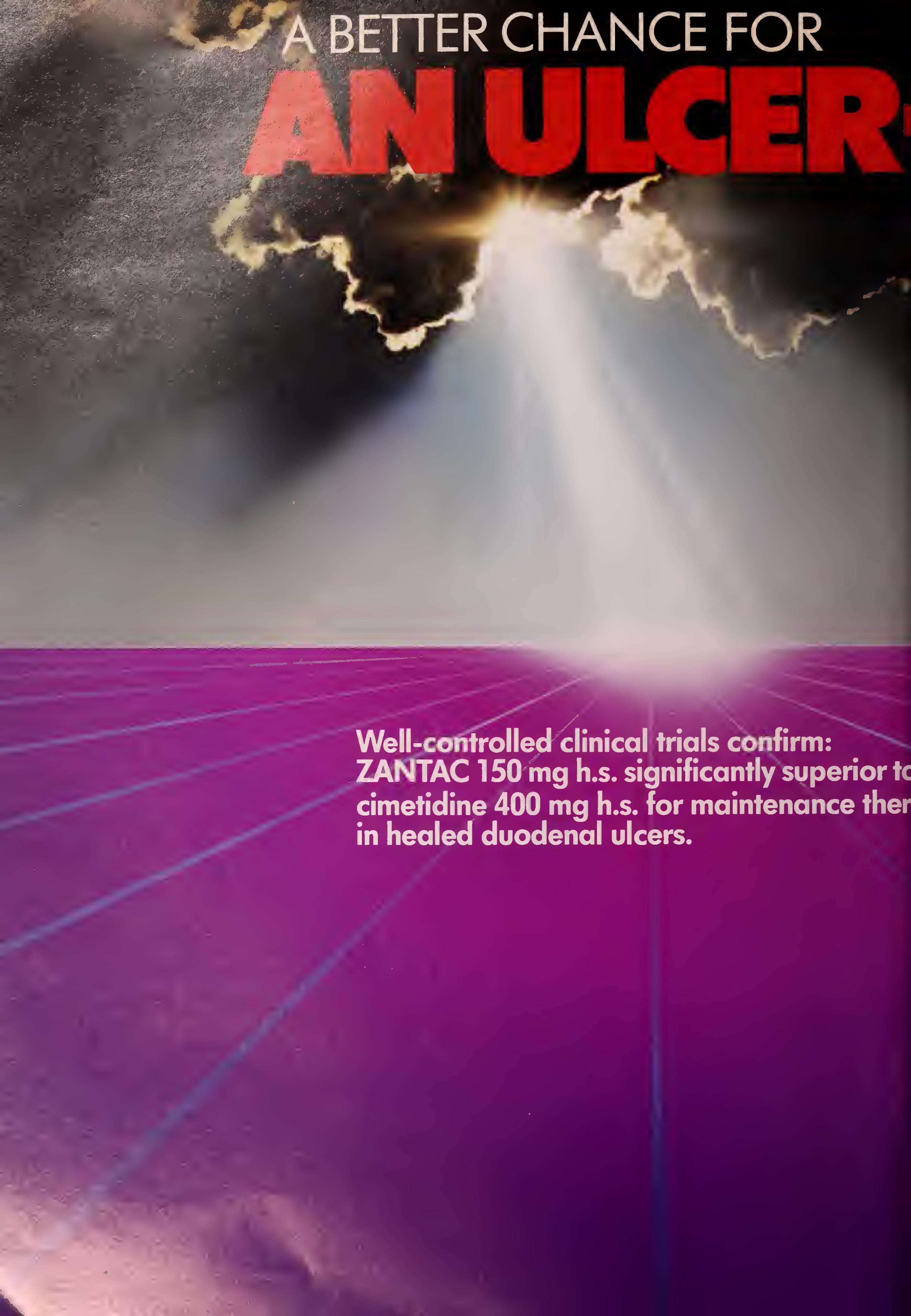

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ZANTAC
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cimetidine
00 mg h.s. (n=241) 63%

.01 †P=0.0004 % life-table estimates

All patients were permitted prn antacids for relief of pain.
Adapted from Silvis[1] and Gough[2]

These two trials[1,2] used the currently recommended dosing regimen of cimetidine (400 mg h.s.) and ranitidine (150 mg h.s.). A comparison of other dosing regimens has not been studied.
The studied dosing regimens are not equivalent with respect to the degree and duration of acid suppression or suppression of nocturnal acid.
The superiority of ranitidine over cimetidine in these trials indicates that the dosing regimen currently recommended for cimetidine is less likely to be as successful in maintenance therapy.

Zantac® 150 *h.s.*
nitidine HCl/Glaxo 150 mg tablets

laxo/ ROCHE® See next page for references and Brief Summary of Product Information.

ZAN375 July 1987

References: 1. Silvis SE, Griffin J, Hardin R, et al: Final report on the United States multicenter trial comparing ranitidine to cimetidine as maintenance therapy following healing of duodenal ulcer. *J Clin Gastroenterol* 1985;7(6):482-487.
2. Gough KR, Korman MG, Bardhan KD, et al: Ranitidine and cimetidine in prevention of duodenal ulcer relapse: A double-blind, randomised, multicentre, comparative trial. *Lancet* 1984;ii:659-662.

BRIEF SUMMARY OF PRODUCT INFORMATION

ZANTAC® 150 Tablets
(ranitidine hydrochloride)
ZANTAC® 300 Tablets
(ranitidine hydrochloride)

The following is a brief summary only. Before prescribing, see complete prescribing information in ZANTAC® product labeling.

INDICATIONS AND USAGE: ZANTAC® is indicated in:
1. Short-term treatment of **active duodenal ulcer.** Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD).** Symptomatic relief commonly occurs within one or two weeks after starting therapy and is maintained throughout a six-week course of therapy.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

CONTRAINDICATIONS: ZANTAC® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS: Symptomatic response to ZANTAC® therapy does not preclude the presence of gastric malignancy.

Since ZANTAC is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see **DOSAGE AND ADMINISTRATION**). Caution should be observed in patients with hepatic dysfunction since ZANTAC is metabolized in the liver.

False-positive tests for urine protein with Multistix® may occur during ZANTAC therapy, and therefore testing with sulfosalicylic acid is recommended.

Although recommended doses of ZANTAC do not inhibit the action of cytochrome P-450 enzymes in the liver, there have been isolated reports of drug interactions which suggest that ZANTAC may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Lack of experience to date precludes recommending ZANTAC for use in children or pregnant patients. Since ZANTAC is secreted in human milk, caution should be exercised when administered to a nursing mother.

ADVERSE REACTIONS: Headache, sometimes severe, seems to be related to ZANTAC® administration. Constipation, diarrhea, nausea/vomiting, and abdominal discomfort/pain have been reported. There have been rare reports of malaise, dizziness, somnolence, insomnia, vertigo, tachycardia, bradycardia, premature ventricular beats, and arthralgias. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients.

In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid IV for seven days, and in 4 of 24 subjects receiving 50 mg qid for five days. With oral administration there have been occasional reports of reversible hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice.

There have been rare reports of reversible leukopenia, granulocytopenia, thrombocytopenia, and pancytopenia.

Although controlled studies have shown no antiandrogenic activity, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving ZANTAC, but the incidence did not differ from that in the general population.

Incidents of rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia, have been reported, as well as rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia) and small increases in serum creatinine.

OVERDOSAGE: Information concerning possible overdosage and its treatment appears in the full prescribing information.

DOSAGE AND ADMINISTRATION: Active Duodenal Ulcer: The current recommended adult oral dosage is 150 mg twice daily. An alternate dosage of 300 mg once daily at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated.

Maintenance Therapy: The current recommended adult oral dosage is 150 mg at bedtime.

Pathological Hypersecretory Conditions (such as Zollinger-Ellison Syndrome): The current recommended adult oral dosage is 150 mg twice a day. In some patients it may be necessary to administer ZANTAC 150-mg doses more frequently. Doses should be adjusted to individual patient needs, and should continue as long as clinically indicated. Doses up to 6 g/day have been employed in patients with severe disease.

Benign Gastric Ulcer: The current recommended adult oral dosage is 150 mg twice a day.

GERD: The current recommended adult oral dosage is 150 mg twice a day.

Dosage Adjustment for Patients with Impaired Renal Function: On the basis of experience with a group of subjects with severely impaired renal function treated with ZANTAC, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

HOW SUPPLIED: ZANTAC® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) and unit dose packs of 100 tablets (NDC 0173-0393-47).

ZANTAC® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 tablets (NDC 0173-0344-42) and unit dose packs of 100 tablets (NDC 0173-0344-47).

Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.

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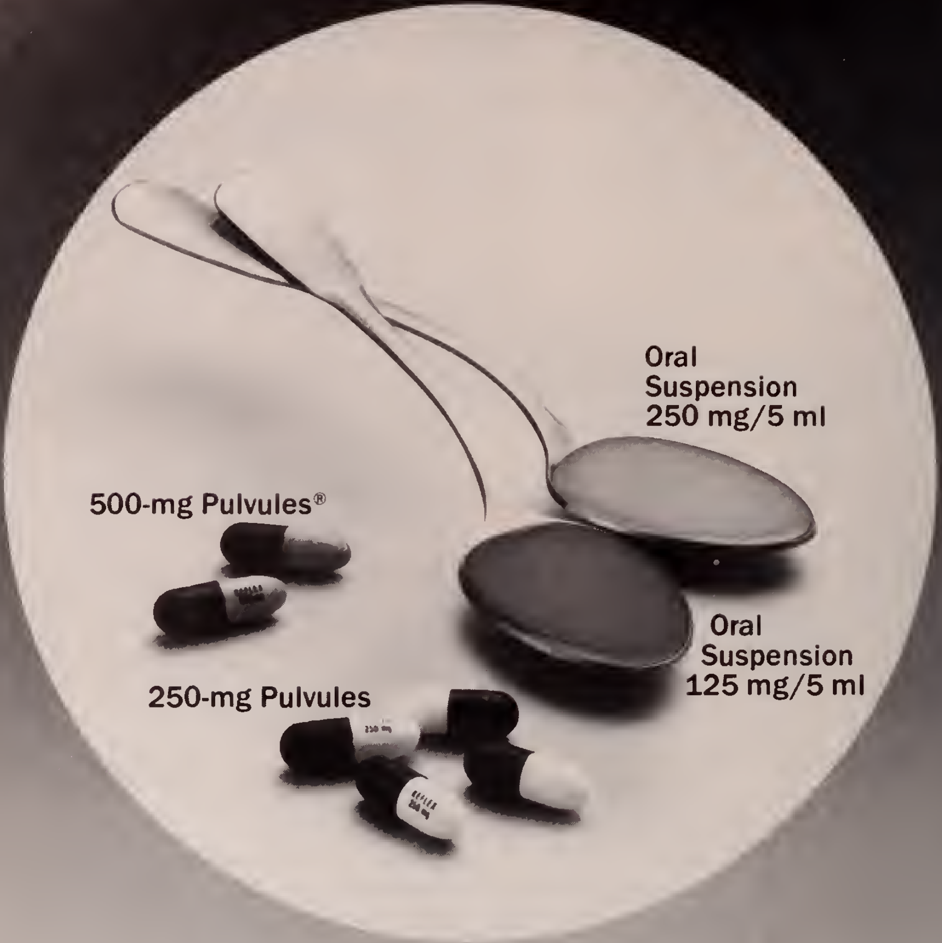

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Executive Privilege

What's in a year anyway? We know it's made up of days and months with holidays thrown in here and there, and we certainly know that the seasons come and go during the year, and most of us even have a birthday during the year. But for me . . . this particular year has been one continuous blur of acronyms and abbreviations, MICA, MIEC, WCCA, HCFA, APHP, DRG, AAFP, ASMA, and the list never seems to end.

As my first year draws to a close, I pensively recall my second week . . . as I sat back in my chair with all the files neatly in order, my pencils sharpened and laying in a row, the desk smelling of lemon polish, and the paper clips deftly in their plastic container. I now felt I was ready for ACTION! So, like any qualified executive, I called the staff into my office and leaned over the new desk pad my wife had recently bestowed on me and stared intently at them . . . but not for long. Soon my authoritarian stare drifted to my old awards garnered from another industry and noticed they were already covered in dust. I hoped this wasn't a portent of things to come. But, I quickly retook control and assertively, asked "OK, what do I do now?" I immediately heard a rhythmic gasp expending in unison and noted a quizzical look of discernment on their faces, knowing that I had made an immediate impression. "Well, what is it I'm suppose to do?" I repeated, finally realizing what they thought I should do. But fortunately for me they displayed complete professionalism and appeared to be sympathetic to a drowning man. It was an auspicious beginning I thought, to a profession that offered succinct answers and demanded a high intellectual capacity. Now I was convinced that the staff thought I was void on both counts. But, I was confident that ASMA would offer more than chest pains, but in many respects I found it was barely breathing.

So, I was hired to breath life into this association, I thought to myself. Without further ado, I set out to do just that. Except . . . how could I ever possibly please 500 bosses when I can't even please the one at home? Do I have the intellectual capacity and steely resolve to resuscitate the association I pondered to myself. No . . . but I plowed ahead anyway. However, by the third week life was easier. Why? Because I was attending the MIEC annual meeting in California. I soon displayed my ignorance when I asked what MIEC stood for. Physicians and peers alike shrugged and moved the conversation elsewhere, so I took up space during the meeting and filled pages of legal pads with information that I read and reread many times. What did it mean? I didn't have a clue. Like a duck out of water, I waddled through many of the issues.

But I'll give them credit, physicians are a patient lot. They spoon fed and eye dropped information into that mental void, allowing me time to digest what I needed and the opportunity to regurgitate the rest. They have provided the freedom to be a duck or a void. In the process the brain began to function, and I've waddled closer to the pond.

I'm thankful for the support of officers and staff, for family and friends, and in particular, . . . this year.

Raymond Schalow
Executive Director
Alaska State Medical Association

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Auxiliary News

Alaska State Medical Association Auxiliary

DO YOU WANT TO BE ON THE OPRAH WINFREY SHOW? That was the invitation Oprah issued to auxilians from fifty states who gathered recently in Chicago for the AMA Auxiliary convention. Carolyn Crouch (a national committee member), Jenny Johnson, and I (representing the state) were among 700 people who listened to speaker Oprah Winfrey. Oprah spoke on self-esteem which was very appropriate. The auxiliary is important, and you are important. With your help, this will be a year to remember.

At the convention there was a great deal of interest in Alaska. Wherever we went, people wanted to talk to us. They were impressed with our programs and the fact that we, a small state auxiliary, had the great successes that we had last year. You can be proud of so many things. Champ Camp was a great success. Children's Healthfest '87 was an exciting event. At the convention, we won two AMA-ERF awards. Yes, you can be proud.

Our new national president, Betty Szewczyk, has long been active in programs for the family. Betty is a good speaker and an exciting leader. She makes you want to go out and do things, and I can't wait to get started. Her theme for the year is TEEN HEALTH. Can you think of some wonderful programs with that in mind?

My goal for the year is increased membership. Medical practice is changing radically, and the auxiliary is in the best possible position to tell the story of medicine. We know what our husbands and wives go through, their day-to-day pressures, and their concern for their patients. We have a big job to do, but we are equal to the task. I challenge every person reading this to get just one new member to join. We can do it. Please send dues of $40.00 today to Aaltje Smith, 4733 Pavalof, Anchorage, Alaska 99507.

Join us and maybe you can be on the Oprah Winfrey Show.

Cassie Hill
President, ASMA Auxiliary

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Editorial

The past two months have been hectic, rewarding, and exciting for arctic science and *Alaska Medicine*. With this issue, *Alaska Medicine* has once again become a bimonthly publication. Trying to meet our shorter deadlines was a challenge for everyone—but we made it! We have received some more superb manuscripts that will be published in future issues—keep your eyes peeled for your next issue!

Providence Hospital has provided an excellent, thought provoking article on some of the many challenges we Alaskans now face as we struggle to provide high quality care to our patients. But Providence Hospital provided much more to *Alaska Medicine* than just an article. Recognizing that excellence in science is an integral part of excellence in patient care, Providence Hospital provided a grant to *Alaska Medicine* to cover the cost of publishing this issue. No scientific publication is able to support itself solely through subscriptions. We are no exception. *Alaska Medicine* can contribute to excellence in medicine and patient care through publication of original manuscripts and feature articles. The journal can become a credible forum to present important information on the issues we must all confront and solve together to assure that our patients will benefit from our cumulative wisdom. I thank Providence Hospital for its extraordinary contribution.

I am delighted to announce that the Alaska Public Health Association voted at its annual meeting in May to adopt *Alaska Medicine* as its official scientific journal. All members of ALPHA will receive *Alaska Medicine* for one year and will have a special opportunity to submit articles for publication in our feature section. We are expanding our readership as well as increasing our role in support of arctic medical science.

Interest in the arctic has soared, reaching heights not achieved since Peary and Cook raced each other to the North Pole in 1909 and since the discovery of vast oil deposits was announced in 1960. Interest in arctic medical research has never been greater. The University of Alaska has negotiated a landmark agreement with the Siberian Branch of the USSR Academy of Medicine to collaborate on arctic health research. The National Academy of Sciences has finished its Five Year Plan for arctic research, including an excellent chapter on health research, according to requirements of the Arctic Research and Policy Act of 1984. The 7th International Congress on Circumpolar Health (ICCH) was held in Umea, Sweden in June, attended by more than 500 health professionals, including 97 Alaskans. The first meeting of the general assembly of the newly formed International Society for Circumpolar Health was held successfully in Umea. And, ASMA enjoyed a superb annual convention in Juneau.

We now can look forward to the many exciting activities that will surround further development of the Siberian—University of Alaska arctic health research program, the 1987 ARCTIC SCIENCE CONFERENCE to be held September 23-26 at the Sheraton Anchorage Hotel, and the annual meeting of the American Society for Circumpolar Health that will be held September 24, 1987 at the Sheraton Anchorage Hotel during the Arctic Science Conference.

This space contributed as a public service.

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Guest Editorial

Alcoholism — A Glimmer of Hope

For those of us involved in health care at any level, the personal and public health consequences of alcoholism are staggering. Alcoholism is a global issue. It does not discriminate on the basis of age, sex, color, or social status. Representative national statistics are overwhelming.

> Alcohol-related problems cost the United States more than $60 billion a year; are responsible for 20 percent of the national expenditure for hospital care; cause the birth of a great many disabled children every year; are involved in more than a third of our suicides, half of the fatal highway accidents, a large number of rapes and sex crimes, two-thirds of the deaths by drowning, 70 percent of deaths by falls, 83 percent of deaths by fire, and a majority of domestic violence episodes.[1]

Alcoholism and its consequences are considered by most to be the number one health problem in the State of Alaska. The high priority given to alcoholism is reflected by the abundant funding of alcohol treatment programs. The State of Alaska's budget for drugs and alcohol for 1987, although cut at midyear, is approximately $10 million. In addition, the Alaska Area Native Health Service will spend another $1.4 million in 1987. Even higher levels of spending have occurred in past years.

The outcome of this effort—modest at best.

Alcoholism and its treatment have been particularly frustrating for health care professionals and the Native community in our State. Is there any hope?

I think there is! The Alkali Lake Indian Tribe of British Columbia has been able during the last 14 years to transform itself from 100% alcoholism to 95% sobriety. Their experience has been dramatized in a video presentation produced and acted by themselves, entitled "With Honor For All." It's a must-see for all those working in the field. Their efforts were not orchestrated by any federal or state program. Change occurred when several alcoholic tribal members became sober, recruited other members of the tribe, and eventually were able to mobilize their community to take a dramatic stand against alcohol.

It was a painful process. It took time. The effort continues.

The extraordinary article on alcohol in this issue was written by Carl Hild, who was adopted by a Native family and had extensive experience living in rural Native Alaska. He presents several theoretical concepts related to the potential root cause of the failure of many of our currently conceived alcoholism programs with some suggestions of what future efforts might take. Of particular importance for western workers are the differences of world view and concepts of communication. Mr. Hild's ideas are thought provoking and useful for all of us who either provide human services or are in program development related to Native programs.

I think there is a glimmer of hope on the horizon. Alcoholism can be conquered as demonstrated at Alkali Lake. There, it has been a difficult but productive 14 years. The time course will not be any shorter nor the effort any easier in the north country.

Thomas S. Nighswander, M.D., M.P.H.
Anchorage, Alaska

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[1] West LJ (ed). Alcoholism and related problems: issues for the American public. Prentice Hall, Englewood Cliffs, CA, 1984, p.209.

Letters to the Editor

Dear Editor:

The event which prompted this letter was a notice from my malpractice carrier that my rates will be increased to approximately $74,000 next year. I'm well aware that for this amount of money I am being represented by a company with an excellent record and a strong financial base of operations. I'm grateful for the protection which this type of policy affords me in my group practice, and I would hope that I am never called upon to practice without it. However, it has occurred to me recently that what I am providing for this premium ought to be called maloccurrence insurance rather than malpractice insurance.

I have never had any difficulty accepting responsibility for my own acts. I would certainly hope to be held liable for any willful act on my part which caused harm to a patient. As we all know, though, there are many events which occur in medicine which are not a direct consequence of physician care such as breakage of orthopedic appliances, some postoperative wound infections, and biological variations in the rate and degree of healing between different patients.

Perhaps it would be more reasonable if physicians and surgeons provided true malpractice insurance for damage caused by errors of omission and willful acts and allowed the patient to secure separate coverage for the other misadventures which may befall a person during his inpatient or outpatient treatment. For instance, a patient could be offered the option through a hospital or a physician's office of purchasing a small supplemental policy to cover the treatment in question. This would be quite similar to policies that have long been available through travel agencies. In this way the patient would be directly assuming the cost for providing coverage for all untoward events which might occur to him outside the scope of true malpractice.

R. W. Garner, M.D.
Anchorage Fracture and Orthopedic Clinic
Anchorage, Alaska

MIEC Claims Prevention Seminars Set For August

ASMA and Medical Insurance Exchange of California (MIEC) are cosponsoring a series of Claims Prevention Seminars in Alaska in August for physicians. A separate series of programs will be provided for medical office staff and hospital allied health professionals. The schedule:

- Monday, Aug 24, 1987

  Providence Hospital, Anchorage
  12 noon "Malpractice Prevention for Medical Assistants and Nurses"
  6:30 p.m. "Malpractice Prevention for Physicians"

- Tuesday, Aug 25, 1987

  Fairbanks Memorial Hospital, Fairbanks
  12 noon "Malpractice Prevention for Medical Assistants and Nurses"
  6:30 p.m. "Malpractice Prevention for Physicians"

- Tuesday, Aug 26, 1987

  Bartlett Memorial Hospital, Juneau
  12 noon "Malpractice Prevention for Medical Assistants and Nurses"
  6:30 p.m. "Malpractice Prevention for Physicians"

The programs for physicians will cover five major liability issues: informed consent, medical record documentation, medication-related malpractice claims, communication problems that lead to injury, and patient education as a deterrent to litigation. The seminars will address some of the nonmedical issues which can complicate and destroy the defense of a medically-defensible lawsuit.

Seminars for medical assistants will discuss the "7 C's of Malpractice Prevention: Competence, Compliance, Charting, Communication, Confidentiality, Courtesy and Carefulness." Continuing medical education credit is being offered for the physician program.

MIEC will host a luncheon at the noontime programs and dinner for the physician seminars. All programs are free of charge to any physician who is a member of ASMA or who is an MIEC insured. Seminars for medical office staff also are provided at no cost. Preregistration is required. To preregister, telephone ASMA, 562-2662.

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Anticholinergic Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract
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(PROPRANOLOL HCl) LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

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NEW LOW DOSE
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Please see next page for brief summary of prescribing information

The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION. SEE PACKAGE CIRCULAR)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride INDERAL LA is available as 60 mg 80 mg 120 mg and 160 mg capsules

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites When access to beta-receptor sites is blocked by INDERAL the chronotropic inotropic and vasodilator responses to beta-adrenergic stimulation are decreased proportionately

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol resulting from the slower rate of absorption of propranolol Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval In most clinical settings however such as hypertension or angina where there is little correlation between plasma levels and clinical effect INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate systolic pressure and rate pressure product INDERAL LA can provide effective beta blockade for a 24-hour period

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension it may be used alone or used in combination with other antihypertensive agents particularly a thiazide diuretic INDERAL LA is not indicated in the management of hypertensive emergencies

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope INDERAL LA also improves exercise performance The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation Clinical improvement may be temporary

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock 2) sinus bradycardia and greater than first-degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL

WARNINGS. CARDIAC FAILURE Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle

ONCE-DAILY

INDERAL® LA

(PROPRANOLOL HCl)

LONG ACTING CAPSULES

60 mg 80 mg 120 mg 160 mg

*

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics and the response observed closely, or INDERAL should be discontinued (gradually if possible)

> IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy Therefore when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema) — PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors

MAJOR SURGERY The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol However, such patients may be subject to protracted severe hypotension Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers

DIABETES AND HYPOGLYCEMIA Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels

THYROTOXICOSIS Beta blockade may mask certain clinical signs of hyperthyroidism Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm Propranolol may change thyroid function tests, increasing T_4 and reverse T_3, and decreasing T_3

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranolol

PRECAUTIONS. GENERAL Propranolol should be used with caution in patients with impaired hepatic or renal function INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test Withdrawal may leac return of increased intraocular pressure

CLINICAL LABORATORY TESTS Elevated blood urea levels in patients with severe h disease elevated serum transaminase alkaline phosphatase lactate dehydrogenase

DRUG INTERACTIONS Patients receiving catecholamine-depleting drugs such as r pine should be closely observed if INDERAL is administered The added catecholan blocking action may produce an excessive reduction of resting sympathetic nervous ac which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthos hypotension

Caution should be exercised when patients receiving a beta blocker are administer calcium-channel-blocking drug especially intravenous verapamil for both agents ma press myocardial contractility or atrioventricular conduction On rare occasions, the conc tant intravenous use of a beta blocker and verapamil has resulted in serious adverse react especially in patients with severe cardiomyopathy congestive heart failure or recent my dial infarction

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol

Ethanol slows the rate of absorption of propranolol

Phenytoin, phenobarbitone and *rifampin* accelerate propranolol clearance

Chlorpromazine, when used concomitantly with propranolol results in increased pl levels of both drugs

Antipyrine and *lidocaine* have reduced clearance when used concomitantly propranolol

Thyroxine may result in a lower than expected T_3 concentration when used concomi with propranolol

Cimetidine decreases the hepatic metabolism of propranolol, delaying eliminatior increasing blood levels

Theophylline clearance is reduced when used concomitantly with propranolol

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY Long-term stud animals have been conducted to evaluate toxic effects and carcinogenic potential month studies in both rats and mice employing doses up to 150 mg/kg/day, there w evidence of significant drug-induced toxicity There were no drug-related tumorigenic e at any of the dosage levels Reproductive studies in animals did not show any impairm fertility that was attributable to the drug

PREGNANCY Pregnancy Category C INDERAL has been shown to be embryoto animal studies at doses about 10 times greater than the maximum recommended human

There are no adequate and well-controlled studies in pregnant women INDERAL shou used during pregnancy only if the potential benefit justifies the potential risk to the fetus

NURSING MOTHERS INDERAL is excreted in human milk Caution should be exer when INDERAL (propranolol HCl) is administered to a nursing woman

PEDIATRIC USE Safety and effectiveness in children have not been established

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have required the withdrawal of therapy

Cardiovascular Bradycardia, congestive heart failure, intensification of AV block, hyp sion, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually Raynaud type

Central Nervous System Light-headedness, mental depression manifested by insc lassitude, weakness, fatigue reversible mental depression progressing to catatonia disturbances, hallucinations vivid dreams, an acute reversible syndrome characteriz disorientation for time and place, shor memory loss, emotional lability, sl clouded sensorium, and decreased p mance on neuropsychometrics For imm formulations, fatigue, lethargy and dreams appear dose related

Gastrointestinal Nausea, vomiting, e tric distress, abdominal cramping, dia constipation, mesenteric arterial throm ischemic colitis

Allergic Pharyngitis and agranuloc erythematous rash, fever combined wit ing and sore throat, laryngospasm and re tory distress

Respiratory Bronchospasm.

Hematologic Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic pu

Auto-Immune In extremely rare instances, systemic lupus erythematosus has reported

Miscellaneous Alopecia LE-like reactions, psoriasiform rashes, dry eyes, male impo and Peyronie's disease have been reported rarely Oculomucocutaneous reactions in the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) ha been associated with propranolol

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochlori sustained-release capsule for administration once daily If patients are switched from IN Tablets to INDERAL LA Capsules, care should be taken to assure that the desired thera effect is maintained INDERAL LA should not be considered a simple mg-for-mg substi INDERAL INDERAL LA has different kinetics and produces lower blood levels Retitrati be necessary, especially to maintain effectiveness at the end of the 24-hour dosing int

HYPERTENSION — *Dosage must be individualized* The usual initial dosage is INDERAL LA once daily whether used alone or added to a diuretic The dosage r increased to 120 mg once daily or higher until adequate blood-pressure control is ac The usual maintenance dosage is 120 to 160 mg once daily In some instances a dosage mg may be required The time needed for full hypertensive response to a given do variable and may range from a few days to several weeks

ANGINA PECTORIS — *Dosage must be individualized* Starting with 80 mg INDEF once daily, dosage should be gradually increased at three- to seven-day intervals until response is obtained Although individual patients may respond at any dosage le average optimal dosage appears to be 160 mg once daily In angina pectoris, the val safety of dosage exceeding 320 mg per day have not been established

If treatment is to be discontinued, reduce dosage gradually over a period of a few wee WARNINGS)

MIGRAINE — *Dosage must be individualized* The initial oral dose is 80 mg INDEF once daily The usual effective dose range is 160-240 mg once daily The dosage increased gradually to achieve optimal migraine prophylaxis If a satisfactory respons obtained within four to six weeks after reaching the maximal dose, INDERAL LA therapy be discontinued It may be advisable to withdraw the drug gradually over a period of weeks

HYPERTROPHIC SUBAORTIC STENOSIS — 80-160 mg INDERAL LA once daily

PEDIATRIC DOSAGE — At this time the data on the use of the drug in this age group ar limited to permit adequate directions for use

*The appearance of these capsules is a registered trademark of Ayerst Laboratorie

REFERENCES:

1. INDERAL LA National Compliance Evaluation Program Data on file, Ayerst Labora
2. Ravid M, Lang R, Jutrin I The relative antihypertensive potency of propranolol, oxp atenolol, and metoprolol given once daily *Arch Intern Med* 1985; 145 1321-1323

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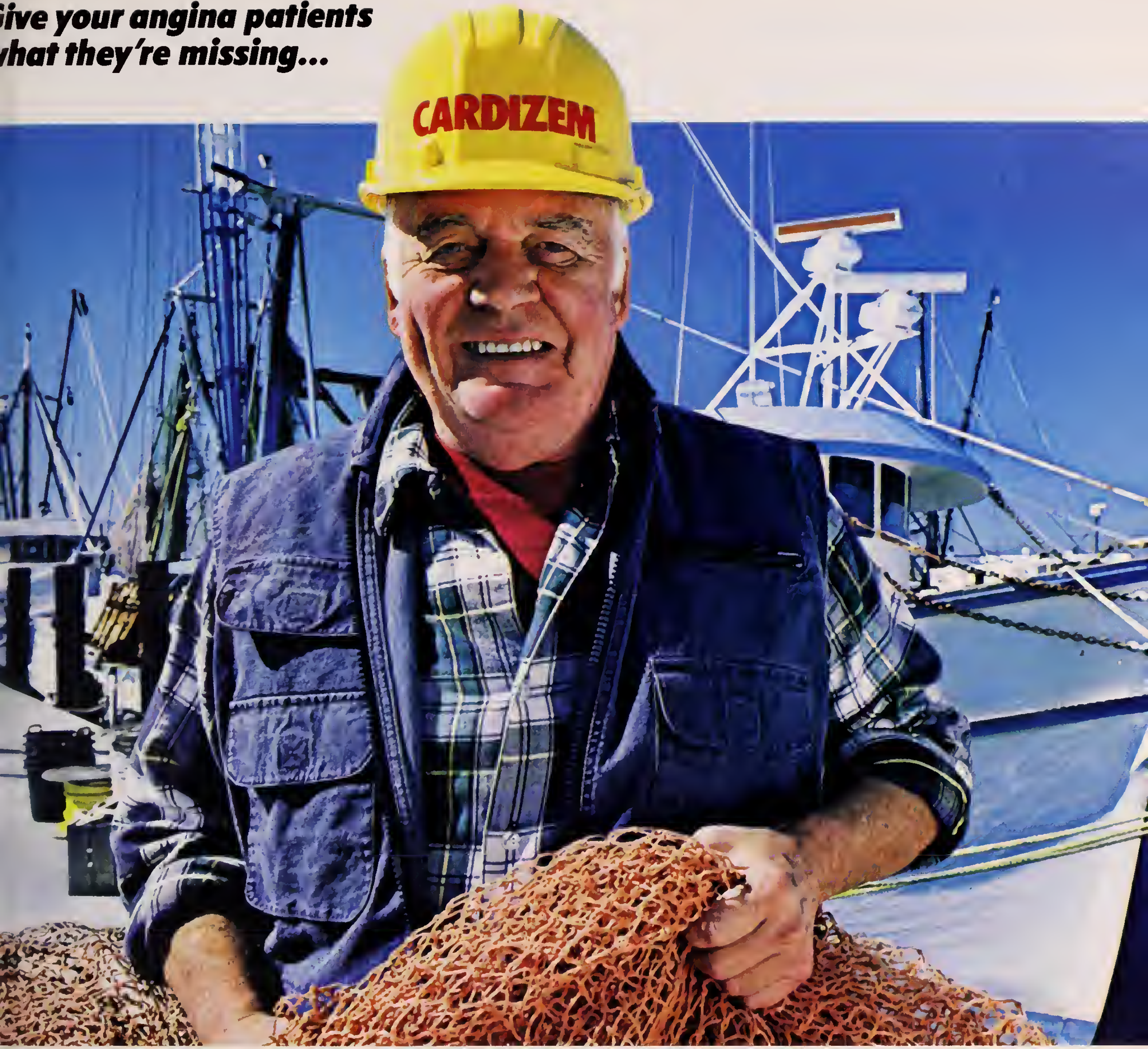
hat they're missing...



CARDIZEM®: FEW SIDE EFFECTS

diltiazem HCl/Marion

- **Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate**
- **Proven efficacy when used alone in angina[1]**
- **Compatible with other antianginals[2,3]***
- **A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]**

90 mg GREATER DOSAGE FLEXIBILITY
NEW 120 mg

***See Warnings and Precautions.**

Please see brief summary of prescribing information on the next page.

CARDIZEM® FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

diltiazem HCl/Marion

60 mg tid or qid

Brief Summary
Professional Use Information

CARDIZEM®
(diltiazem HCl)
30 mg, 60 mg, 90 mg, and 120 mg Tablets

CONTRAINDICATIONS

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

WARNINGS

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)

PRECAUTIONS

General. CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

Drug Interaction. Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

Carcinogenesis, Mutagenesis, Impairment of Fertility. A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

Pregnancy. Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women; therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers. Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.

Pediatric Use. Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

Cardiovascular:	Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.
Nervous System:	Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.
Gastrointestinal:	Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.
Dermatologic:	Petechiae, pruritus, photosensitivity, urticaria.
Other:	Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.
Issued 9/86

See complete Professional Use Information before prescribing.

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Human Injury from Bear Attacks in Alaska, 1900–1985

by John P. Middaugh, M.D.[1]

ABSTRACT

During 1900–85, 105 people were documented to have been injured, 20 fatally, by bears in Alaska. Although hunters accounted for 49% of those injured during 1900–79, campers (31%) and hikers (35%) accounted for the majority of those injured 1980–85. The number of persons injured has increased in recent years. Attacks appeared to be associated with risk factors previously described, and current recommendations to avoid or reduce injuries were substantiated by this study. Increased public education and research efforts are warranted to ensure safe coexistence of humans and bears in Alaska.

INTRODUCTION

The drama of human encounters with grizzly *(Ursus arctos horribilis)* and black *(Ursus americanus)* bears is both frightening and fascinating. Man's increasing encroachment into previously remote, rugged bear habitat has confronted park managers and wildlife biologists with increasing difficulties in safeguarding both people and bears (1–4).

Yet the literature contains few well documented studies of human injuries from bear attacks (5,6). Between 1872–1980, Herrero (7) documented 130 human injuries associated with 111 incidents involving grizzly bears in the national parks. During the same period, Herrero reported that at least 165 people were injured in 143 incidents involving grizzly bears. In comparison, human injuries associated with encounters with black bears were more frequent. Between 1960-80, more than 500 injuries were documented from national parks, only 3 of which involved major injury. Outside of national parks, at least 23 people were killed by black bears between 1900 and 1980 (7).

Human injuries from bear attacks in Alaska have increased in recent years. Bear and human management strategies must be implemented now, taking advantage of the knowledge gained through the successes and failures of past management strategies. Accurate information on bear encounters must be obtained and used to direct efforts at areas where risks are highest and to identify risk factors amenable to intervention.

METHODS

Incidents of human encounters with bear that resulted in human injury were compiled from books (7–9), magazines (10–13), newspaper articles (14–16), and reports available through the National Park Service and through the Alaska Department of Fish and Game. Only incidents where contemporary documentation was available were used in this study. In many cases, incidents could be verified from more than one source. Incidents where bear encounters were documented but in which human injury did not occur were omitted from this study.

RESULTS

During 1900–85, 105 people were documented to have been injured by polar bears, black bears, and grizzly bears in Alaska. Only two were known to be Native Alaskans. There were 20 fatalities. During the past decade, the number of humans injured has steadily increased (Figure 1).

Of 105 individuals injured, 19 (18.1%) were injured by black bears, including 4 fatal injuries; 85 (81.0%) were injured by grizzly bears, including 16 fatalities; and 1 (0.9%) person was injured by a polar bear. The case-fatality rate for those attacked by black bears was 21% and by grizzly bears was 18.8%.

Between 1900–85, 43 (41.0%) of the 105 people injured were hunting, 19 (18.1%) were hiking, 12 (11.4%) were camping, and smaller numbers were engaged in trapping, mining, logging, fishing, geological exploration, photography, and unknown activities. However, a major change occurred in the activities of those injured in recent years. Between 1900–79, of 79 humans injured, 39 (49.4%) were hunters, followed by injuries to 10 (12.7%) hikers and four (5.1%) campers. During 1980-85, however, of the 26 individuals injured, only four (15.4%) were hunters while nine (34.6%) were hikers and eight (30.8%) were campers (Figure 2). The number of hunters injured per year did not increase in the past decade.

Of 25 incidents involving a grizzly sow with cubs, one (4.2%) resulted in a fatality compared to 15 (25.0%) fatalities from 60 incidents involving a solitary grizzly bear. Most of the encounters with grizzly sows and cubs were abrupt and unexpected. Attacks were brief (seconds to minutes). After the sow neutralized the perceived threat, the bears departed. In only one incident

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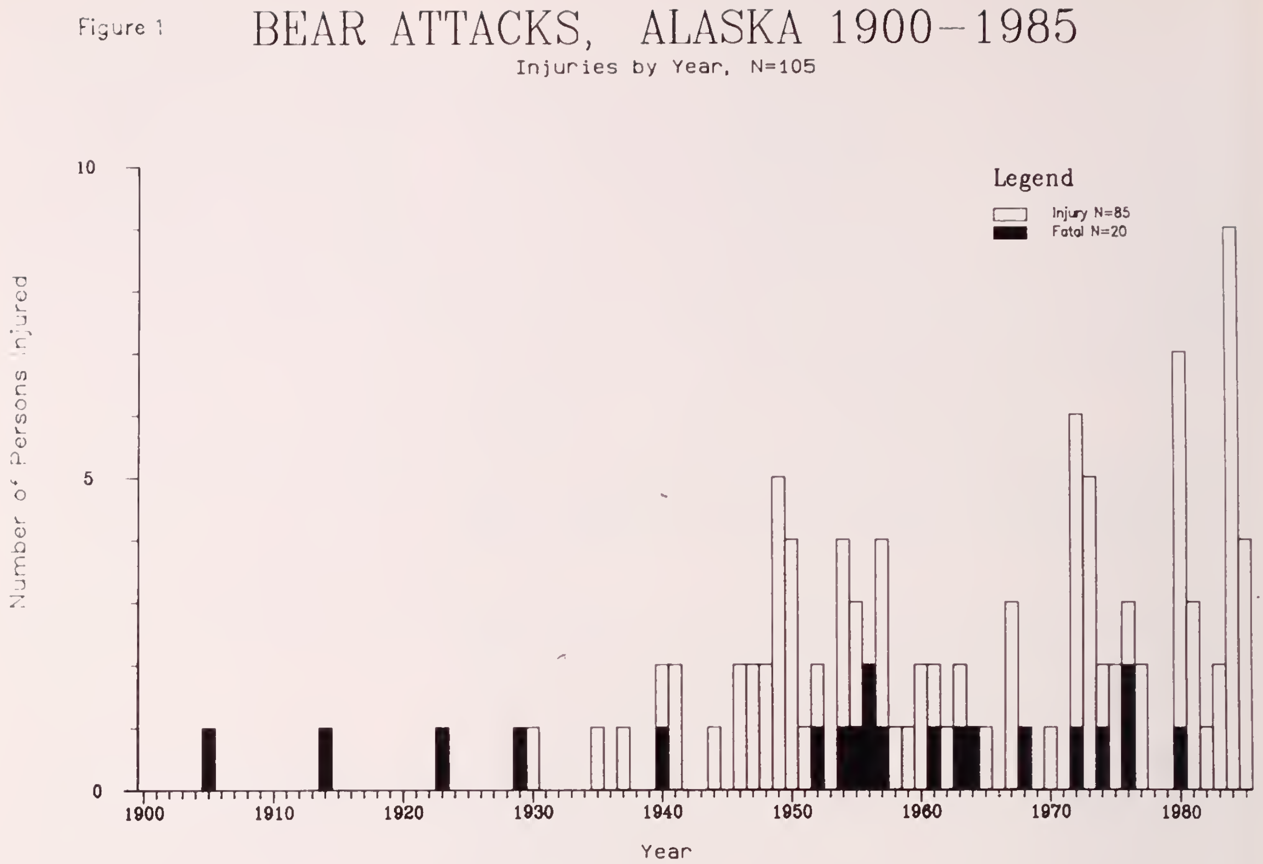
[1] Epidemiology Office, Division of Public Health
Department of Health and Social Services
State of Alaska

Figure 1


BEAR ATTACKS, ALASKA 1900–1985

Injuries by Year, N=105

did a second attack involving a sow and cubs occur.

In most instances only one person was injured in an attack, and in no cases were more than three humans injured in any single attack. In three incidents, two grizzly bears attacked one person; all other attacks were inflicted by a single bear (Table 1). Most attacks occurred during summer months and during the fall hunting season (Figure 3).

Of the 43 hunters injured in 40 attacks, only one was injured by a black bear. In only 12 of the 40 attacks were data recorded about the nature of the attack. In these 12 attacks, hunters were charged at close distance with little warning; in three of these 12 attacks, the bear previously had been wounded by the victim. In 17 of the 40 attacks on hunters, data were available to indicate that the bear was shot and killed, wounded, or driven off by the victim or his companions. In one instance, however, a severe injury suffered by a victim resulted from a bullet wound — a companion shot at the attacking bear but instead struck the person being attacked.

Of the 31 incidents involving a camper or hiker, in only two were data recorded to document that the victim successfully drove off the bear by using a firearm. No data were available on the number of hikers or campers who were carrying firearms at the time of the attack. In four other attacks on three fisherman and one photographer, the victim or his companions killed, wounded or drove off the attacking bear with firearms. In four attacks on individuals engaged in other activities, the victim carried a firearm, but the attack took place so quickly that the firearm was not able to be used.

Black bear attacks resulted in four fatalities and three severe maulings, all solitary attacks. Details of the four fatal attacks are not available. Only minor injuries were sustained by 12 other individuals who fought and made noise in the remaining eight attacks, driving away the attacking bear.

In nine incidents involving grizzlies and six incidents involving black bears, the injured person was not attacked until he or she turned and ran away. In four instances, grizzly bears attacked a second time when the victim moved shortly after the initial attack apparently had ceased. After an attack was imminent or had begun, nine people played dead — eight during grizzly attacks and one during a black bear attack. The person attacked by the black bear was alone and sustained severe injuries before being rescued.

Among 12 individuals who were known to be without firearms, nine attacked by a grizzly bear and three attacked by a black bear fought back. All survived. Other individuals who yelled, made noise with firecrackers or by firing a gun in the air found the attack continued in two instances involving grizzlies and found the attack broken off in four instances involving grizzlies and two instances involving black bears.

Severe facial injuries were suffered by 11 people; 10

Figure 2 BEAR ATTACKS, ALASKA

by Activity of Victim

1900-1979 (N=79)

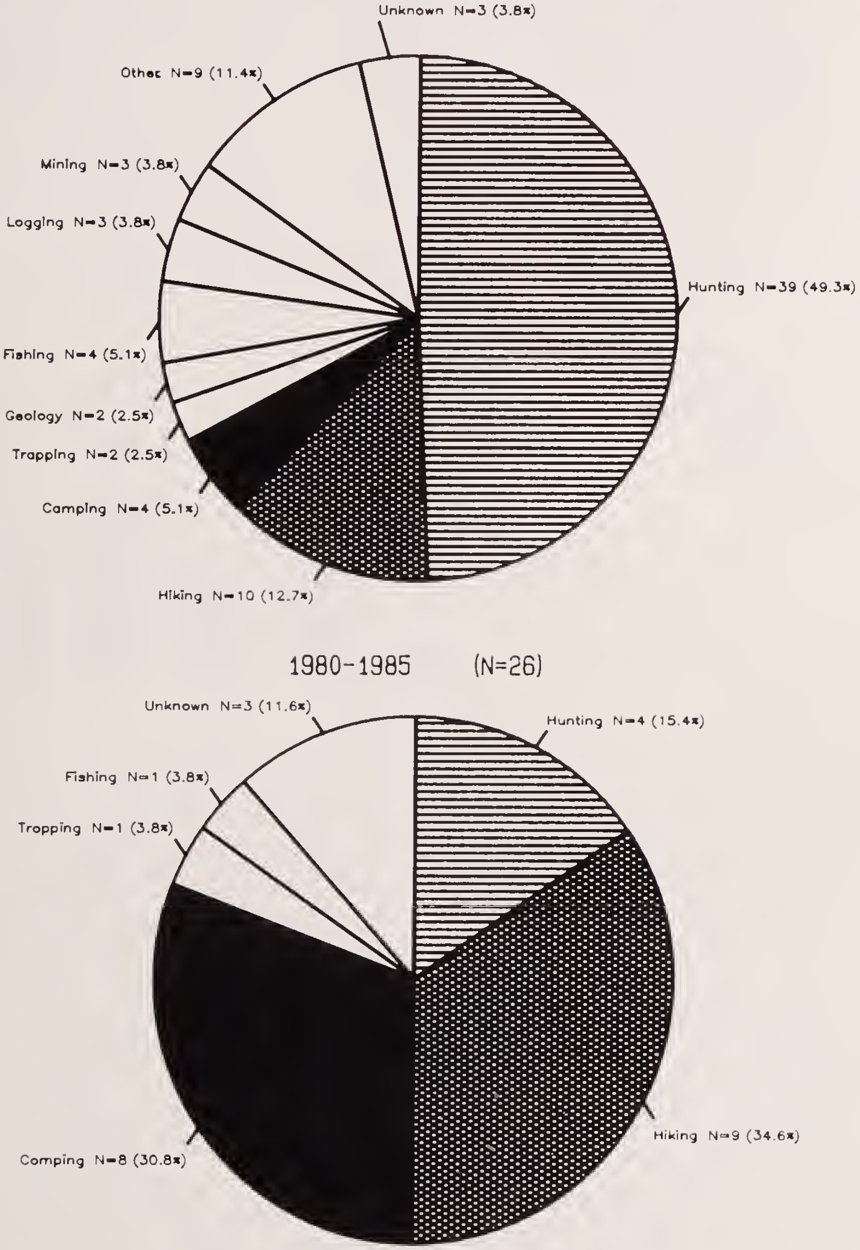


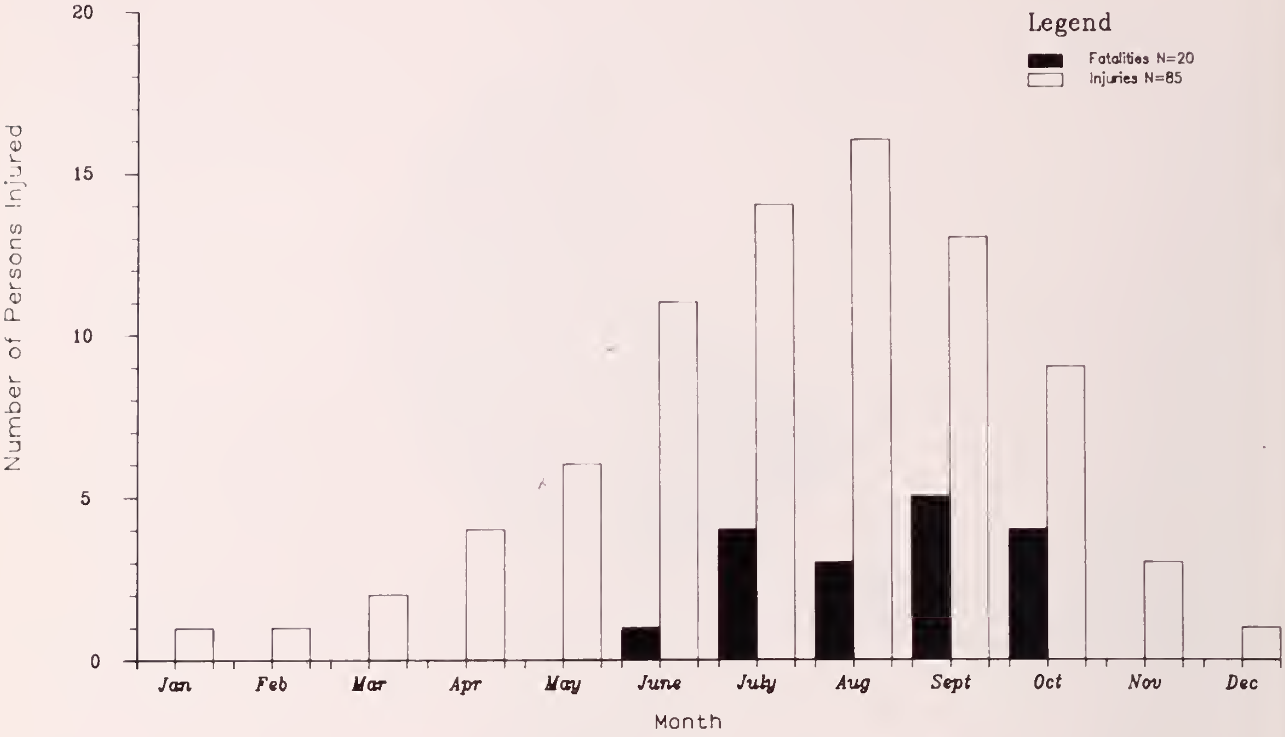
Table 1. BEAR ATTACKS, ALASKA, 1900-1985

NUMBER INJURED PER ATTACK	NUMBER OF ATTACKS				NUMBER OF PEOPLE INJURED			
	Polar	Grizzly	Black	Total	Polar	Grizzly	Black	Total
1	1	71*	12	84	1	71	12	84
2	0	7	2	9	0	14	4	18
3	0	0	1	1	0	0	3	3
TOTAL	1	78	15	94	1	85	19	105

***In three cases, one person was attacked by two grizzly bears.**

Figure 3 BEAR ATTACKS, ALASKA 1900–1985

Injuries by Month, (N=105, Unknown=7)



were from grizzly bears. In 12 other incidents in which the victim protected the face by lying down or by curling up in a ball, the bear bit the skull, tearing off pieces of scalp, but caused no skull fractures. Skull fractures were documented in two cases and may have occurred in three others, all fatal.

Hikers and campers were injured in different geographic locations than hunters. Hunters were injured in four major areas; Southeast (Admiralty Island), Kenai Peninsula, Kodiak Island, and the interior near Fairbanks (Figure 4). Of 31 hikers and campers injured, 10 were injured in Denali National Park. Human activity increased dramatically in Denali Park between 1975-85 with recreational visits rising from 160,600 in 1975 to 436,542 in 1985 (15). Between 1980–85, seven injuries occurred in Denali Park among hikers and campers during a time when 1,972,545 recreational visits were counted for an average rate of 3.5 injuries per one million recreational visits.

DISCUSSION

Between 1975 and 1985, Alaska's population doubled, and tourism increased substantially. Prior to 1980, almost one-half of individuals injured by bears in Alaska were hunters. However, after 1980, hikers and campers accounted for 65% of those injured, and seven of 17 hikers and campers injured were injured in Denali National Park. While it is possible that increased reporting and case ascertainment accounted for the apparent increase, the number of hunting related injuries remained constant, suggesting that the observed increase was real and primarily due to an increase of injuries among campers and hikers.

Of the 19 individuals injured by black bears, four (21%) were killed. Although grizzly bears are known for their ferocity and the seriousness of injuries they inflict in human attacks, 16 (18.8%) of 85 individuals injured by grizzly bears died. The similar case-fatality rate among those injured by black bears and grizzly bears is quite different from that reported by others outside Alaska. For example, Herrero reported only three major injuries out of more than 500 injuries inflicted by black bears in North American national parks between 1960–80 (7).

Fatalities were infrequent in attacks by a grizzly sow with cubs. These attacks are believed to almost always be defensive and usually result from an abrupt and unexpected encounter (7). When the person threatening the cubs has been dealt with by the sow, the bears depart. The brief duration of these encounters supports the conclusion that the attacks were defensive, triggered by the sow's need to protect her cubs.

Severe injuries occurred from facial bites. Since grizzly bears often grab each other about the jaws, bears may instinctively attempt to attack the face. Because of the irregularity of facial bony structures, easy purchase

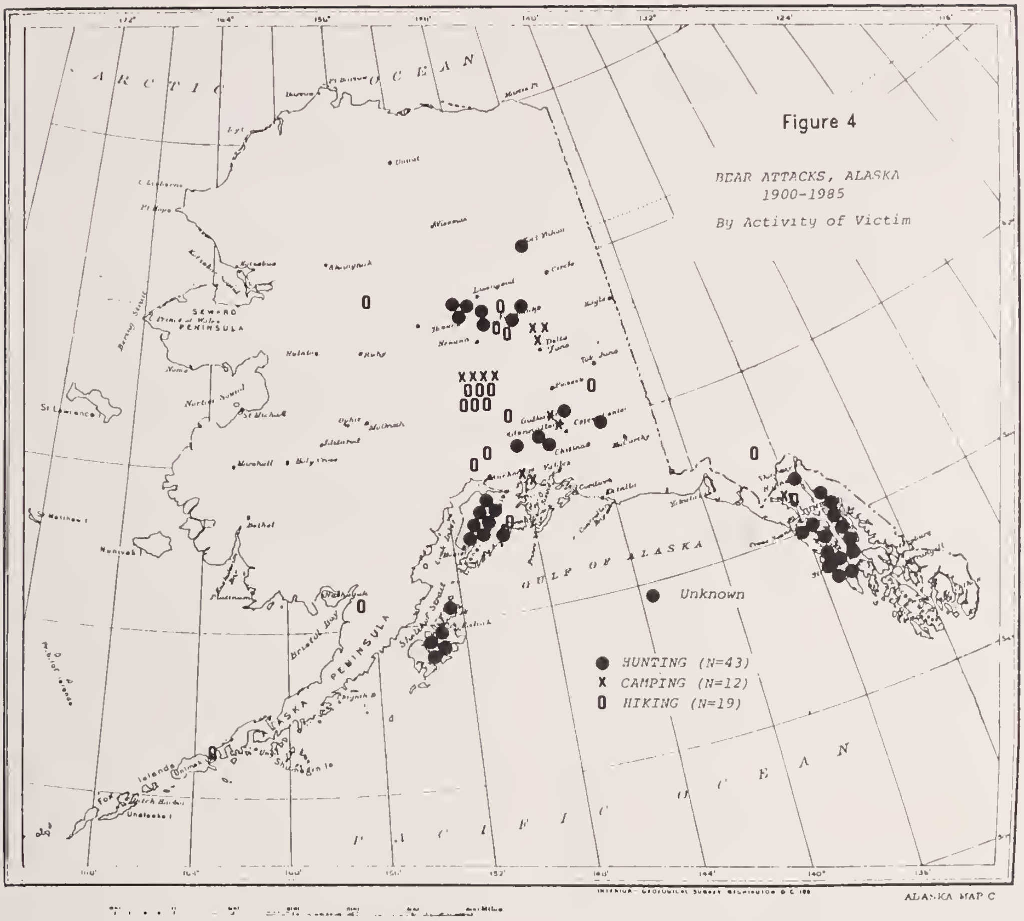


Figure 4

BEAR ATTACKS, ALASKA
1900-1985
By Activity of Victim

is available for bear's teeth and severe injuries may result. In contrast, many individuals bitten on the back of the head sustained extensive soft-tissue scalp injuries, but skull fractures were rare. If attacked by a grizzly, every effort should be made to protect the face. Studies are needed to determine the safest position to assume to avoid fatal injuries during attacks.

Herrero has recommended that different measures be taken by potential victims depending on the attacking species and circumstances of the attack. Passive behavior is recommended only for defensive attacks by grizzly bears. If the attack is an offensive attack by either a grizzly bear or a black bear, then escape or fighting back is indicated. A person never should play dead during a serious attack by a black bear (7).

Herrero's recommendations to back country enthusiasts to prevent injury require a very cool head and clear thinking by the potential victim. Exceptional courage is required *not* to run when charged by a grizzly bear, to play dead when appropriate during an attack, and to remain motionless for some time after the attack ends. Yet, his recommendations appear to be sound based upon examination of reported attacks in Alaska. Several victims who unexpectedly encountered a bear were not charged or attacked until they turned and ran. In four grizzly attacks, the initial attack was brief and injuries inflicted were relatively minor. The victim, believing that the attack was over, moved or got up only to be attacked again with greater ferocity and resultant increased severity of injuries.

Severe injuries were sustained by one woman, attacked by a black bear while alone, who played dead. When the seriousness of the attack became evident, vigorous resistance might have reduced her injuries. More serious injuries and probable death were avoided by a man who vigorously fought a black bear that attacked without warning.

The size of the party appears to be important. Larger groups may be more intimidating to the bear or may make more noise. Hunters, who make less noise than campers and hikers and often hunt alone or widely separated from partners, on several occasions were charged by bears in dense underbrush, at close distances, and with little warning.

There were only two documented reports in which a camper or hiker successfully fended off a bear by use of a firearm, particularly surprising because many campers and hikers in Alaska carry firearms. In contrast, hunters were frequently able to shoot the attacking animal. In at least one instance involving hunters, however, the victim's most severe injury resulted from a bullet wound when a companion shot at the attacking bear but instead struck the person being attacked. In four cases the attack was so sudden that the victim was unable to bring the firearm into use. Few data are available on encounters that did not result in injury, on the numbers or proportions of campers and hikers who carried firearms, or on instances where firearm use prevented an attack. Reports of incidents where firearms were successfully used to reduce human injury occurred primarily among hunters. From the limited data available, it appears unlikely that firearms would be of benefit in reducing injuries among hikers and campers.

Only two Alaskan Natives are known to have been injured by bears. It is unlikely that underreporting of injuries, particularly in the past two decades, can account for this finding. More information is needed to determine the reasons, but knowledge of the land, bear habitats and behavior, less reluctance to shoot the animal, and less recreational invasion of bear territory may explain the difference.

In spite of the great public concern about bear attacks, no system exists in Alaska to collect systematically information about human-bear encounters or injuries. Data are scant. Encounters that do not result in human injury are rarely, if ever, reported. Data presented in this study must be interpreted with caution. Information about attacks was incomplete, not systematically collected at the time of the incident, and available for only a small proportion of the documented encounters. Denominator data are lacking and little, if any, data about many of the attacks were available. Analyses were, of necessity, limited to the incidents where detailed data were reported.

Apart from Herrero's work, little information is available to determine risk factors associated with encounters resulting in injury compared to encounters without injury. Data on species, age, sex, and presence of cubs of attacking bears also are of poor quality. No single agency is responsible for investigating human-bear encounters. A sentinel injury reporting and surveillance system to document information on bear encounters and associated injuries may provide valuable information in the future.

In comparison to other risks, the risk of human injury from bears is extremely small — 3.5 injuries per million recreational visits in Denali Park between 1980–85. The risk to back country users is higher but difficult to quantify because of the lack of accurate data on the number of people at risk. In comparison to the 20 fatalities caused by bears between 1900–85, 20 Alaskans were killed by dogs between 1955-85 (17); and during 1983–84, 20 Alaskans were killed in crashes associated with three-wheeled, all-terrain vehicles (18).

Wildlife managers and national park administrators must prepare for more human-bear encounters in Alaskan parks and in Alaskan wilderness. Increasing efforts to educate the public about risk reduction behavior is warranted. Improved surveillance and incident investigation is needed to collect accurate data, to define risk factors, and to evaluate current recommendations so that we can ensure the safe coexistence of humans and bears in Alaska in the future.

ACKNOWLEDGMENTS

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Yersiniosis in Alaskan Children: Case Reports and Discussion of Clinical and Epidemiological Variability

by Kenneth W. Moss, M.D.[1]

ABSTRACT

Yersinia enterocolitica causes a wide variety of clinical syndromes including diarrhea and enterocolitis, pseudo-appendicitis, septicemia, and arthritis.

This study reports four cases seen in Juneau, illustrating the wide variety of clinical presentation. Severity ranged from a mild illness to severe pseudo-appendicitis and a moribund state. Treatment was with chloramphenicol, TMP-SMX, or supportive care only, and all recovered without sequelae.

Yersinia enterocolitica was seldom diagnosed in comparison to other causes of bacterial enteritis. The first Alaska case was reported in 1981, with a high number of 13 cases in 1984, and 4 cases in 1985 and 1986.

It is yet unclear whether Yersiniosis occurs less frequently in Alaska than elsewhere or whether it occurs without being identified, because isolation of Yersinia requires special media or techniques.

Health care providers should be aware of it as an etiology of these syndromes, specifically culture for it when appropriate symptoms occur, and report diagnosed cases promptly.

INTRODUCTION

Yersinia enterocolitica causes a wide variety of clinical syndromes including diarrhea and enterocolitis, pseudo-appendicitis, and arthritis (1–5). More severe illness includes hepatitis, carditis, meningitis, Reiter's syndrome, septicemia, and rashes including erythema nodosum (5–7). The gamut of presentation ranges from a nuisance illness to a confusing diagnostic problem (8) with attendant risk of multiple diagnostic studies. In the most severe form of illness, a case-fatality rate of 50%, despite antibiotic treatment has been reported (2,9,15).

Yersinia infection has been identified as a cause of diarrheal disease more frequently in the 1970s and 1980s (2,7,11,12). It has been long recognized as a pathogen in European countries but only recently so in North America. The incidence in Eastern Canada is high compared to that in the U.S. and Western Canada (1,4,7).

The infection seems to occur more frequently in cooler climates than in warmer ones. *Y. enterocolitica* is unique among the Enterobacteriaceae in a number of ways, most notably in the ability to multiply at low temperatures and in the temperature dependence of biochemical reactions and motility. Although the organism is widespread, most of the reports have come from countries with cooler climates such as Canada, Scandinavia and other northern European countries, rather than tropical climates. For example in a large hospital medical center in the Midwest (Cincinnati) the 12-month occurrence of Yersinia was only 3 cases, compared to 59 for Salmonella, 41 for Campylobacter, and 14 for Shigella (20). The first case reported in Alaska was in 1981; since then, increasing numbers have been reported in epidemiological reviews (13,14).

We hereby report four cases of *Y. enterocolitica* infection in children from Southeast Alaska to draw attention to the variable clinical presentation.

CASE REPORTS

Case I: A five-year old white male was seen on several occasions for proctitis. He had only slightly loose stools over a three-day period, but irritation of the rectal area remained in spite of attention to and treatment for pinworms, food allergies, and a negative search for giardiasis. He was afebrile and had no cramps. A search for pathogens isolated *Y. entercolitica* from stool at the time he was spontaneously improving.

Case II: An 18-month old white male was admitted from his home in a logging camp residence because of fever to 102° F (41° C) for five days and four or five episodes of nonbloody diarrhea per day for five days. He was cheerful and fairly well hydrated, though febrile. His WBC was 32,700 with 31% bands on admission. He was treated with I.M. penicillin-G and with trimethoprim—sulfamethoxazole (TMP-SMX) after blood and stool cultures were obtained. He had rapid improvement from his illness, and *Y. enterocolitica* was identified from stool the day after his discharge from the hospital.

Case III: A four-month old American Indian male was brought from his home village of Hoonah with history of fever to 105° F (40.5° C) and diarrhea for two days. Watery, green diarrhea had persisted in spite of dietary restrictions and oral rehydration. He was lethargic, mildly dehydrated, and febrile. His WBC was 23,700 with 60% PMNs and 15% bands. Blood, urine and CSF cultures showed no growth. Consideration for sepsis and for appendicitis was given and consultation obtained. He was thought not to have a surgical abdomen; chloramphenicol and ampicillin were given pending culture results. The patient responded well, became afebrile, and his WBC dropped to 12,100 with 35% PMNs, 1% band. *Y. enterocolitica* was identified from stool culture after the patient was discharged from the hospital, improved.

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Case IV: A three-year old white male from a homestead farm near Petersburg, Alaska was admitted because of repeated vomiting and diarrhea. He had been mildly ill with abdominal pain, vomiting and diarrhea 10 days prior to his admission, and he was thought to have a viral illness. He improved transiently with symptomatic and supportive care. On the day of admission, however, his physician noted that he was much more ill, febrile and dehydrated; his WBC was 33,800 with 59% PMNs, 27% bands. He appeared tired and listless, dehydrated, febrile to 104.0° F (40° C), and had abdominal distention. Abdominal x-rays showed no signs of intestinal obstruction, but gross dilation of the bowel was present. The surgical consultant felt he had no need for surgical exploration and advised rehydration and further studies. After studies of CSF, urine, blood, and stool were obtained, he was given cephapirin intravenously and later, chloramphenicol. He remained ill; repeat WBC was 35,000, again with a left shift. Initial cultures of the stools were negative for pathogens. However, Yersinia culture techniques were not used on the initial specimens. Repeated stool cultures specifically for Yersinia revealed the organism. He was beginning to feel better and was afebrile on the sixth day of hospitalization with intravenous antibiotics when *Y. enterocolitica* was identified. At this point, the I.V. antibiotics were discontinued and he was given p.o. TMP-SMX. He recovered uneventfully.

DISCUSSION

These cases represent some of the variable presentations of *Y. enterocolitica* infection. "To the microbiologist, *Yersinia enterocolitica* spells charisma; to the patient, it spells trouble; but to the physician, it inevitably spells confusion (3)." Case I, though giving the patient symptoms troublesome enough to seek medical attention, was little more than an inconvenience or nuisance, the organism being almost serendipitously found. Case II represents a sicker patient in that the child had fever, signs and symptoms enough to warrant hospitalization. Children under five-years old are said to be much more inclined to have a self-limited enteritis only; older children and adults are more likely to have terminal ileitis or the "pseudo-appendicitis" picture (1,2,5,7).

The third category involves systemic disease, typically in adults, characterized by bacteremia, polyarthritis, arthralgia, Reiter's syndrome or erythema nodosum (2,7,17,18), and is not relevant to the cases discussed. Cases III and IV, though in small children, do illustrate the perplexing dilemma of an acutely ill child who appears septic and who may "call forth the surgeon's blade" (3). Appendectomies have been performed, only to find an anatomically non-inflamed appendix but a later recovered organism (1,2,7,8). Examination of the CSF, blood, and blood cultures may be appropriate; and x-rays, ultrasonography or nucleide scans may be warranted in the search for the cause of the illness. Though the hospitalized cases reported here all had uncomfortable and severe illness and alarmingly elevated blood counts, none had septicemia or the erythema nodosum or arthritis frequently described in Scandinavian literature (6,7,15,17,18). VanTrappen (7) describes symptoms in 37 patients having:

Abdominal pain	84%
Diarrhea	78%
Fever	43%
Anorexia	22%
Weight loss	16%
Nausea	13%
Vomiting	8%

Though the infants and children reported here are too young to describe their symptoms, they seem to fit this listing of signs and symptoms.

Recovery of the organism from feces may be difficult, not because of the lack of growth, but because of the overgrowth of normal flora. Indeed, Yersinia may be camouflaged initially by a more rapidly growing fecal flora and may take longer to be isolated and identified. The ability of *Y. enterocolitica* to grow at low temperature (4° C - 25° C) offers a means of isolating it from other gram negative bacteria. It is motile at 25° C, but not at 37° C. At times cold enrichment techniques are necessary for its isolation. It is generally a lactose non-fermenter.

Diagnosis is possible by titration of antibodies in the patient's serum, which rise after six days of illness, peak at 3-4 weeks of illness, and may last for several months (7). Serological diagnosis was developed and is used in Europe, but is not commonly available in North America. *Yersinia enterocolitica* can be biochemically separated into 5 biotypes and by serologic techniques into 34 serotypes. Some serotypes such as 0:3, 0:8, and 0:9 are isolated frequently from human sources, while others (0:1, 0:2, 0:16) appear to be animal strains. Serotypes 0:3 and 0:9 predominate in France, Scandinavia, Japan, South Africa and Canada. These two serotypes are unusual in the USA where type 0:8 predominates (3).

Several plating media have been proposed for directly isolating *Y. enterocolitica* (21-23). One of the selective media, CIN agar, was first described by Scheimann, in 1979, and employs cefsulodin, Irgasan, novobiocin, bile salts and crystal violet as the selective agents (21). The agar base inhibits the growth of gram positive and a number of gram negative organisms.

The differential property is based on fermentation of mannitol. Organisms capable of fermenting mannitol produce a localized pH drop around the colony which imparts a red color to the colony.

The epidemiology of Yersiniosis remains somewhat unclear. It may be endemic to an area (1,2,5,7) or epidemic in nature, as evidenced by outbreaks (2,8,16). Reservoirs of infection have been both domestic and wild animals, food and milk, or waterpower, as documented by the above reports. There seems to be a definite predilection for cool climates.

Epidemiological departments should consider making Yersiniosis a reportable disease along with other enteric infections. Studies and reports will tell more clearly the geographically epidemiological pattern and will also help to tell if Yersiniosis is indeed the same infection, or if bacteria are serologically or biochemically different in different areas. [*Yersinia enterocolitica* infections are reportable in Alaska. Editor's note]

Clinicians need to be aware of this bacterium as a source of illness and culture specifically for it. This is especially important if the presentation indicates consideration of surgical exploration. Specific diagnosis will give the patient or parent (as well as the health practitioner) satisfaction of specific diagnosis and appropriate antimicrobial therapy may shorten the course of the illness and may reduce or eliminate possible unnecessary diagnostic studies.

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Prevalence of Enteric Pathogens in Two Alaska Day Care Centers

by Sue Anne Jenkerson, M.S.N., R.N.C.[1]
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ABSTRACT

The prevalence rate of enteric pathogens in the absence of reported disease was studied in two arbitrarily selected Alaska day care centers. Stool samples from 59 children were examined for enteric bacteria, parasites, and viruses. Of 59 children tested, eight (14%) cases of giardia were identified; further investigation found associated illness in three families. No other parasites or pathogenic bacteria were found. From 21 children, 25 viruses were isolated, a prevalence rate of 42%. Rotavirus, recovered from 11 children, was the most common viral isolate. Application of public health measures will reduce morbidity and costs among day care center attendees and their families.

INTRODUCTION

Diarrheal illness in day care centers is a growing concern for public health officials. Outbreaks of diarrhea in day care centers caused by shigella (1), campylobacter (2), giardia (3), cryptosporidium (4), rotavirus (5), and hepatitis A (6) are well documented. Outbreaks of salmonella in day care centers are less frequently reported but have been documented (7).

Several investigators have determined prevalence rates of enteric pathogens in day care centers in the absence of known illness. Surveys in Houston day care centers showed prevalence rates of giardia to be 21% and 26% (8), and of rotavirus to be 12.4% (6). Day care centers in Hampton County, South Carolina were shown to have a prevalence rate of giardia of 26% (9). We examined the prevalence rates of common enteric pathogens in two day care centers in Anchorage, Alaska as part of a larger ongoing study of enteric pathogens in Alaska.

METHODS

Nurses were stationed at two arbitrarily selected day care centers during each day of one week in January 1986. No cases of enteric disease had been reported from either day care in recent months. No cases of diarrhea or fever among day care children or staff were reported to the nurses at the start of, or during the week by either staff or parents.

Medical histories were not obtained on day care attendees prior to collecting stool samples. No attempt was made to gather clinical information from parents until laboratory results were available. Parents of children positive for giardia were then contacted and interviewed. No attempt was made to follow up children positive for viruses, since viral culture results were not reported until six weeks after the study.

Stool samples were collected from each participant and divided into three portions: Carey–Blair transport medium for bacterial pathogens; 10% formalin/ether for ova and parasites; and viral transport medium. Our goal was to collect at least three specimens from each participant during the five-day period. Stool samples for bacteria and parasite testing were delivered to state laboratories for processing at the end of each day or kept refrigerated until delivery the next morning. Stool samples for viral cultures were iced until delivered to the laboratory for freezing to –70° F. They were then packed in dry ice and mailed to the processing laboratory, experiencing only one freeze–thaw cycle.

Samples from Carey–Blair transport medium were plated onto Campylobacter blood agar, GN enrichment broth, MacConkey agar, Selenite enrichment broth, Triple Sugar–Iron, and Xylose-lysine–dyoxycholate (XLD) agar for isolation of *Campylobacter, Enterotoxigenic E coli, Salmonella,* or *Shigella.* Samples in formalin/ether were microscopically evaluated for *Cryptosporidium, Dientamoeba fragilis, Entamoeba histolytica, Giardia lamblia,* nematodes, trematodes, and cestodes.

Viral samples were inoculated into both Rhesus monkey kidney (RMK) and human diploid (M7) cell cultures and incubated at 37° C. Cultures were read on a daily basis and positive cultures showing development of cytopathogenic effect (CPE) were harvested for identification. Routine cell observations were continued for each specimen for 14 days; questionable cells were blind passed into fresh cells. All isolates with enteroviral CPE were identified via Lim–Benyish Melnick intersecting pools. Adenovirus isolates were identified with monospecific antisera via standard neutralization tests. All stool specimens were screened for rotavirus via enzyme-linked immunosorbant assay.

In 1986, 175 day care centers with a capacity of 8,361

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spaces were licensed to operate in Alaska. Although the majority were located in Anchorage (N = 65, 37%), 39 other towns and villages had at least one licensed day care. Additionally, 518 private day care homes with a capacity of 2,784 spaces were licensed across the state.

RESULTS

Stool samples were collected from 59 (42%) of 141 children, five years old or younger, who attended day care on a regular basis; 36 (43%) of 82 in day care A and 24 (41%) of 59 in day care B. Because response rates did not vary meaningfully between the two day care centers, we combined the data. Of those tested, 21 (36%) submitted at least three stool specimens. We tested 85% of children less than two years of age. However, as attendees increased in age, the number submitting stool samples dropped to 15–50% (Table 1).

Table 1. Number of attendees submitting at least one stool by age.

Age (months)	Number Tested	Number Attending Day Cares	%
0–6	2	2	100
7–12	12	14	86
13–18	7	9	78
19–24	14	16	88
25–30	6	12	50
31–36	3	10	30
37–48	5	34	15
49–60	10	44	23
Total	59	141	42

Table 2. Laboratory Positives Among 59 Children Screened at 2 Alaska Day Cares.

Age (mo)	Number Tested	Bacteria	Parasites*	Viruses#
0–6	7	0	0	2
7–12	7	0	0	7
13–18	10	0	2	5
19–24	11	0	1	4
25–30	6	0	2	1
31–36	3	0	0	0
>36	15	0	3	2
Total	59	0	8	25

***All parasite positives were giardia.**
#Viral positives included the following: Adenovirus 1 (5), Adenovirus 2 (4), Rotavirus (11), Polio 3 (4), and unspecified entervirous (1). Four attendees were co-infected with 2 viruses.

Table 3. Laboratory Positives by Sex (N = 59).

	Bacteria	Parasites*	Viruses	Total
Male (N = 33)	0	2	8	10
Female (N = 26)	0	6	13	19
Total	0	8	21	29

***All parasite positives were giardia.**

No pathogenic bacteria were found (Table 2). The only parasite identified was giardia; eight cases, six female and two male, were discovered (Table 3). Of the eight giardia cases, five were in toddlers (children between 13 and 30 months of age, usually in diapers, but walking). Giardia was not found in children less than one year of age (Table 2). In day care A, the six cases of giardia were clustered in two rooms: three cases in the toddler area and three cases in the three- and four-year olds. In day care B, the two cases of giardia were also among children in the toddler room. Of the eight giardia cases, four, including three with symptoms, were also coinfested with rotavirus.

Of the eight giardia cases, six (75%) had symptoms of intermittent diarrhea, gas, bloating, stomach aches, or increased frequency of stools. The duration of symptoms ranged from three weeks to more than one year. Associated family illness was found in three cases. One individual, a five-month old male, previously evaluated for failure-to-thrive and milk allergy, began gaining weight after treatment for giardia. Follow up stools from seven of the eight cases of giardia were negative after treatment, and their symptoms resolved.

Twenty-five viral isolates were identified from 21 participants (Table 2). Rotavirus was the most common viral isolate (N = 11, 44%); Adenovirus 1 and 2 and Poliovirus 3 were also isolated. The prevalence rate for viral isolates among the 59 tested was 42% in day care B (Table 2). Prevalence rates for rotavirus among those tested were 20% in day care A and 17% in day care B. Viral isolates were found in children from less than six months of age through greater than three years of age (Table 2). Of the viral cases, 13 were female, and eight were male (Table 3). Four children, including one with giardia, were coinfected with two viruses.

DISCUSSION

The prevalence rate for giarda (14%) in the two Anchorage day care centers was lower than prevalence rates reported from other studies (21–26%) (8,9). Follow up of identified giardia cases discovered symptomatic, diarrheal illness of long duration in five cases and illness compatible with giardia in three families. The sixth symptomatic case reported acute diarrhea of 3 weeks duration. Illness in these children was not recognized by the day care center staff or even by the parents. The childrens' symptoms resolved after treatment. One child, who had been treated for failure-to-thrive (FTT) and placed on an elimination diet, gained weight after treatment for giardia. Neither parent nor physician had traced the child's FTT to giardia.

Three symptomatic giardia cases were coinfected with rotavirus. We could not determine which agent caused the diarrhea since both giardia (11) and rotavirus (10) can be asymptomatic infections. However, two of these three cases reported duration of diarrhea for over two months, a finding more consistent with giardia than rotavirus (10,11). During past investigations of day care center outbreaks of diarrheal illness in Alaska, we have not found high rates of coexisting pathogens nor have they been described in published reports of day care center outbreaks. The consistent finding of giardia in day care centers has led some authors to recommend no treatment for asymptomatic cases (8,12). The Child Day Care Infectious Disease Study Group, Centers for Disease Control, reserved recommendations for treatment of asymptomatic giardia cases until further studies evaluate the immediate and long-term impact of such treatment (11). However, untreated individuals may pass infectious giardia cysts for several months and often spread the infection to family members (11).

In our study, all eight identified giardia cases, including asymptomatics, were excreting cysts, the infectious form of giardia (11). Six of these eight cases were actually shown to be symptomatic on questioning the parents. Associated illness was discovered in three families. Treatment with appropriate medication is 90% efficacious, decreases the reservoir of infection, and limits likelihood of spread both at the day care center and at home.

Rotavirus, the single most important cause of childhood gastroenteritis (10), was the most prevalent isolate in our study. The prevalence rate of rotavirus (17.6%) was slightly higher than that seen in a survey of 30 day care centers in Houston (12.4%) (6). Our finding of a high prevalence rate of viral infection of both day care centers warrants further study. Because of the limitations involved in this study we were unable to follow up children infected with rotavirus to determine if they had symptoms at the time they were sampled. Studies of viral transmission of enteric diseases in day care centers may shed light on ways to prevent spread of all enteric disease in the day care center setting.

Our study determined the prevalence of enteric pathogens in two arbitrarily-selected day care centers without known illness. The limited scope of the study necessitates caution in interpretation of the findings. We did not collect information on the socioeconomic status of the day care participants. We collected no information to establish the background prevalence of enteric pathogens among children who do not attend day care.

Data on seasonal prevalence of giardia in Alaska are not well established. However, data available from the Epidemiology Office, collected since giardia became a reportable disease in 1984, do not suggest a marked seasonal variation in reported cases. Finally, only one follow-up stool specimen was obtained from the children who were infected with giardia after treatment. In spite of these limitations, our findings are valuable when considered in the context of current understanding and research findings about giardia and other enteric pathogens in children attending day care centers (13).

For the past two years, we have aggressively investigated day care illness, treating both symptomatic and asymptomatic cases of treatable diseases. We established measures to improve disease reporting and routinely interviewed all cases of enteric disease reported to our office. We investigated day care centers linked to any known case, looking for compatible illness. We treated all cases found at the day care centers and among family members including asymptomatics. Aggressive intervention into enteric disease outbreaks in Alaskan day care centers appear to have limited transmission of enteric diseases. Investigations have provided focused opportunities to emphasize handwashing and to detect infected children and adults.

Increased attention has already been focused on the diarrhea dilemma in day care centers (1,13). As more children enter day care, the risk of fecal-oral spread of enteric pathogens increases. Children in diapers are efficient transmitters of enteric pathogens both to other children in the day care and their non-day care contacts and adult family members.

Once a case is reported to the health department, efforts need to be directed at case finding among family members and other young children in the day care, identification of both symptomatic and asymptomatic infected contacts, and consistent treatment of infected individuals. Educating day care center staff to become attuned to detecting and reporting diarrheal illness should be pursued. Day cares need to develop measures (sickrooms, cohorting) to encourage parents to report diarrheal illness instead of concealing illness to prevent their sick child from being excluded from day care. The importance of hand washing by day care center staff after diapering and before meal preparation has been repeatedly shown and cannot be overemphasized (15). These control measures should be coupled with stringent licensing requirements, aggressive enforcement of regulations, continued education of center staff concerning environmental safety and hygiene, and heightened surveillance and reporting of diarrheal illness.

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Free Radicals, the Vitamins A, and Cancer

by Jay E. Caldwell, M.D, M.P.H., F.A.C.S.M.[1]

Cancer is an enigmatic spectrum of diseases which causes in excess of 300,000 deaths annually. The fundamental defect appears to be one of abnormal cellular differentiation. Loss of control is a crucial factor.

Aging, in a sense, is the obverse of cancer. Cells, rather than getting out of control in the proliferative sense, just atrophy. They gradually lose their ability to respond to the vicissitudes of life. This, too, then is loss of control. In aging we cannot turn up the gain control, in cancer we cannot turn it down.

The concept of free radical-induced cellular damage is an exciting and unifying, though doubtfully complete explanation for cancer and aging, as well as for a variety of other problems, such as chronic inflammatory diseases, that have as a common feature, loss of cellular control (1–3).

Cells derive energy by extracting it from the electrons incorporated in carbohydrate and lipid fuels. These reactions occur within mitochondria, the electrons passing down the respiratory enzyme cascade, until adenosine triphospate (ATP) is generated.

The problem with this highly effective system is that it is like playing with fire. Electrons can be very nasty creatures. One may attach itself to a nearby oxygen molecule forming a superoxide. Superoxide is itself highly reactive and may pluck loose an electron from another molecule. The outcome can be general molecular havoc.

Free radicals can react rapidly with cellular, intracellular, and nuclear membranes. A biological "meltdown" can occur in which almost any molecule can have electrons stripped or added (4). More importantly, core genetic material, including a variety of regulatory genes, can undergo mutation.

There was a scare in the fitness world several years ago when a report was published by a group at UC Berkeley (5) documenting the increased free radical and singlet oxygen production induced by exercise in rats. Lost in the usual furor over whether rats were reasonable experimental surrogates for humans was the real gist of the report.

Increased free radical production caused a breakdown of a variety of intracellular organelles, including mitochondria. This served as the prerequisite stimulus for their augmented regeneration. The Berkeley group also observed that endurance training induced an increase in the overall protective antioxidant enzyme system. They suggested that one mechanism for endurance training effects may be free radical action.

The relationship between exercise and malignancy has been recently reviewed (6), the conclusion drawn being that there are simply too many confounding variables to paint a definitive picture. The greatest risk appears to lie with the increased exposure of athletes to toxic components of the environment, such as ultraviolet radiation.

There are a variety of free radical scavengers. Some are enzymes, the most common of which, superoxide dismutase, deactivates superoxide. Hydrogen peroxide and the hydroxyl radical are neutralized by a group of enzymes known as the catalase/glutathione system, integral components of which are the trace minerals, zinc and selenium.

Because there are dozens of possible free radicals, we need a more nonselective sump than these enzymes. Vitamins B1, B6, C, E, and in particular, the A family, play very active roles in quenching this fire.

One of the roles of the vitamins A is the encouragement of cells to mature into their genetically-programmed final form (7). This has important implications for cancer induction. For a cancer to take root, normal cells must first be malignantly transformed. The next step is for the transformed cell to, in fact, reproduce. Not all transformed cells necessarily do this. The first step is called initiation, the second promotion (8).

When there is adequate vitamin A (retinol) activity, cells can differentiate into their fully mature form. Vitamin A appears to be able to force even cancerously transformed cells to differentiate fully, thus rendering them harmless. There is both experimental and epidemiological evidence that cancers derived from epithelial cells are suppressed or prevented by retinoids, which includes both natural and synthetic compounds of the vitamin A family (7).

Carotene is the major dietary source of vitamin A. It is one of a group of naturally-occuring yellowish plant pigments (carotenoids) that scavenge the free radicals generated wherever photosynthesis occurs. The brilliance of the green cholorphyll in the protoplast eclipses the carotenoids' yellow, which only comes to prominence with the death of a leaf in the autumn.

These pigments are responsible for the yellow, orange and red coloration of the peels and pulp of a variety of fruits and vegetables, as well as of the skin of some fish and the exoskeletons of shellfish. Not all carotenoids can be rendered into vitamin A-active compounds, yet all are antioxidants and most seem to have anti-cancer properties.

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Thus, there are two complimentary mechanisms which have been proposed for the anti-cancer effects of the vitamin A family. The carotenoids, of which beta-carotene is the most potent, but including the non-retinol precursors xanthophyll, cryptoxanthin, canthaxanthin, and lycopene, are antioxidants which retard the free radical-induced initiation of malignant transformation of cells. Vitamin A itself inhibits the promotion phase of cancer induction by driving epithelial cells to complete their differentiation (9).

Studies of the relationship between diet and cancer suggest that in populations in which there is adequate vitamin A intake, as in the United States, there is only a weak protective effect for those foods rich in pre-formed vitamin A. The opposite is true in populations with marginal or deficient A intake. A protective effect for the carotenoids, on the other hand, is seen in most populations because of their antioxidant properties (9).

It has been estimated that 35% (30% to 40% in men; 50% to 60% in women) of cancers are diet-related (10). This should not be construed to mean that 35% of cancer is CAUSED by diet, only that diet has an influence in 35%. If free radicals play an important role in cancer cell transformation, it is enticing to believe that the cure for cancer would be to consume more antioxidants.

One could argue that we need more antioxidants than are contained normally in our food because free radical exposure has increased dramatically with the advent of nuclear power and air pollution. Evolution did not plan on this. There are also those who argue that food preparation removes antioxidants from our food resources (11,12).

Laboratory animal studies and human epidemiological analyses do support the theoretical precepts of antioxidant protection against free radical damage. Whether greater than our normal intake is protective is not clear because the risk:benefit ratios for the nutrient antioxidants have not been clarified (10).

Although there are safety margins for vitamins E and C, vitamin A is clearly a dangerous character and shouldn't be monkeyed around with. Regular intake just five times the RDA has been toxic (13).

Beta-carotene, the most potent of the naturally occurring carotenoids appears to be essentially free of toxicity. When excess carotene is consumed we may end up colored like a squash, but we avert vitamin A toxicity by converting only what we need and storing the rest.

For this reason several supplementation trials are underway to observe beta-carotene's effect on cancers of the intestine, lung, and skin. The Physicians's Health Trail presently has 20,000 doctors, among whom I am not numbered, enrolled in a long-term study of prevention of all cancers by providing them with 30mg of beta-carotene daily, about five times the RDA (14,15).

Carotene is the predominant carotenoid found in cantaloupe, cherries, carrots, pumpkin, spinach, sweet potatoes, and squash. Apples, avocados, oranges, pears, strawberries, tomatoes, watermelon, green peppers, and lettuce are major sources of the non-vitamin A variety of carotenoids. Peaches, pink grapefruit, and broccoli have about equal amounts of each variety.

The message for ourselves and our patients is clear. Eat fruits and vegetables. Eschew pills.

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Humana: Proud to be Part of Alaska

We at Humana Hospital – Alaska are very proud to sponsor this issue of *Alaska Medicine* because it provides us the opportunity to communicate what we've accomplished and allows us to express what we see as a continuing and growing need for a strong relationship between the hospitals and physicians.

Though I am a new citizen of the state and have been the hospital's Executive Director for the last five months, I have been involved with Humana Hospital - Alaska for more than five years. During this time, I have always been impressed by the readiness of Alaskans to respond to competition and their ability to pioneer solutions to difficult problems. These are factors we must rely upon to provide a delivery system in Alaska that meets the needs of such a tremendously large state. Focusing on our common interests, we are proud of a healthcare delivery system that is responsive and of unequaled quality.

Due to the tremendous land area, combined with many small outlying communities, delivering quality health care presents unique challenges. In my short time here, I have been able to visit several communities outside of Anchorage and have been impressed with the hospitals and the commitment of physicians in these areas. Physicians and hospitals in this great state have every right to be proud of their capabilities. I also look forward to visiting many other areas and becoming more knowledgeable about health care needs of all Alaskans.

Humana Hospital — Alaska just completed its fifth full year in Alaska. This serves as an appropriate time to reflect on several of our accomplishments during that time.

- DaySurgery was opened in 1983 projecting the increase in outpatient surgery. This unit is separate and distinct, yet offers the convenience of outpatient surgery with the backup of a full service hospital. To complement this service we started the 23-hour observation unit to better meet the individual needs of physicians and patients.
- The first MRI in Alaska was installed on the Humana campus in late 1984 through an arrangement with a group of local physicians.
- The new tower addition in 1986 culminated over four years of technological improvement, expansion, and renovations to the facility.
- Medical office space has been enlarged and currently accommodates close to 100 physicians on our campus.
- The Air Ambulance program provides direct contact with our physician-staffed Emergency Department and transports patients needing additional care from outlying areas. This program, which has a commitment to quality and economical transport, has been very well received throughout the state.
- A 24-hour, in-house anesthesia service was implemented in response to physicians' requests and patients' needs.

Humana has a long term commitment and makes a substantial financial contribution to Alaska. As a proprietary hospital, we receive no state or federal government funds for hospital operations or capital needs except for the patients to whom services are provided. In fact, in fiscal year 1987 we paid more than $2.4 million in local and state taxes. In addition, Humana Hospital - Alaska has shown its willingness to play a vital role providing care to all Alaskans. In fiscal year 1987 more than 18.2 percent of our revenue was written off as unreimbursed care. This is up from 16 percent in fiscal year 1986.

Humana Hospital - Alaska is the 14th largest employer in the state with an annual payroll of more than $16 million.

While we are very proud of our past, we will not rest on our laurels. We will continue to build upon these efforts and work with the physicians to improve and ensure that the delivery system meets the needs of Alaska's future. I am very positive concerning Alaska. Clearly, Alaska has always had wide cyclical swings, each one leading to greater heights. With our great natural and human resources, our prospects are unlimited.

Again, the people who make Humana one of the nation's and Alaska's leaders in health care, are proud and excited about the opportunities of the future here in the Great Land.

George Schneider

George Schneider
Executive Director
Humana Hospital - Alaska

Humana is proud to be part of Alaska. The professionals at Humana Hospital–Alaska are here for a very special reason — to make the good life better, and more healthful, for all Alaskans.

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History of Medicine in Alaska

JAMES ANDREW LUNDQUIST, M.D.

Once upon a time the public schools in the United States were superb, with excellent teachers and a curriculum stressing the humanities and the sciences. Such were the Minneapolis Public Schools when Jim Lundquist was a student. The school in his neighborhood was unusual since its principal, whose wife owned the Kotex Company, put his salary back to be used for enriching purposes. He once bought a tennis court for his school, followed by the football stadium and running track. He made moving pictures of every student.

Jim Lundquist describes the next period of his life as one of wandering. After four quarters spent at the University of Minnesota, he found himself in the military which sent him in turn to the University of Nebraska, Virginia Polytechnic Institute and Princeton University. The emphasis was on engineering, but all engineering credits could be transferred to premed. Jim never did combine his numerous credits and consequently lacks an undergraduate degree.

Jim was on active duty for two years and ten months, from June 1943 until April 1946. He served in the medical department at Fort Totten, N.Y., as a surgical technician. Discharged from the Army, he entered Cornell University Medical College in New York City, receiving his M.D. in 1949. This was an important year in his life since during his internship at Swedish Hospital, Minneapolis he was to marry Alice. Alice had been in junior and senior high school with him and had attended the Minnesota School of Business.

At Swedish Hospital, Jim had a rotating internship, concentrating heavily upon surgery, for which he also had a lengthy preceptorship. He has been an advocate of short courses which provided special training in a number of disciplines related to surgery. At Cornell, he had special studies in orthopedics, ear, nose and throat as well as plastic surgery. After his internship, he practiced with his father for less than a year. Having driven 18,000 miles in this time, he felt ready for another situation, but he wished it to be in a small town with a cool climate.

On May 2, 1951 he flew to Fairbanks on Pan American Airlines. This was to be it. However, his name remained on his father's office door for many years.

Jim Lundquist was to enter a busy and varied practice. His basic discipline was general surgery, including plastic surgery. For years he would do eight or nine major surgeries per week and deliver thirty or forty babies a month. He recalls one day when he saw one hundred and forty-one patients: forty-five in the hospital, ninety-six in the office.

It must be remembered that in the early days of the fifties physicians were thrown upon their own abilities in many situations. Given a good background and a knack for the mechanics of surgery, a doctor performed many procedures from necessity rather than experience or training. It was not easy to transfer patients. There was only one plane a day. Since the trip took eight and one-half hours, there was the problem of survival for patients who needed special procedures.

Consequently, the doctors in Fairbanks often performed operations only read of in a textbook and depended upon manual skill and intuition to perform the necessary. Much general surgery was done using procedures not part of formal education.

Jim Lundquist remembers standing at the windows of the sun porch of the old St. Joseph's Hospital and seeing an airplane hit telephone wires, cartwheeling. The two in the plane were severely injured. One had a subdural hematoma. There were no neurosurgeons, so Jim bore burr holes and evacuated the hematoma. Wryly he remarked: "One learns fast that way."

In 1952, he was the third physician in the U.S. to put tubes in ears. He made his own tubes from polyethylene, creating a flange and finishing it over a soot-free flame. He made tubes daily. Jim was to "do" many tubes in ears and still does.

In the early 70's he made extensive repairs on a man who had been caught in the tail rotor of a helicopter. The man had lost part of his hair, eyelid and eyebrow. Approximately 14 – 15 sq. cms. was missing. Using extended flaps, he constructed a new eyelid and brow.

He cites the dryness of Fairbanks as the reason for an unusually low post-op infection rate. In 1958, he made a study and found that Fairbanks had 1/8th the incidence of infections in all surgeries than in Swedish Hospital, Seattle; 1/20th of that of one hospital in Washington, D.C..

In the thirty-five years he has been in Fairbanks, he has seen the change from general practice to multiple specialists. He has watched his own practice go from mainly surgery and OB to general medicine. He still does ear, nose and throat.

Jim says that his years in Fairbanks were a wonderful time to live because he got to know so many people who were the history of interior Alaska. There were unusual patients too. Jeanie Laurence, widow of Sydney, and herself a painter, was one.

One of his patients had a history of killing three husbands by poisoning. She threatened to kill her fourth. She was a cook, so borrowing one of the 16-inch knives from the kitchen, she pinned him to the bed. She was charged with murder but convicted of involuntary manslaughter. She was sentenced to eight years in a women's prison in West Virginia, but she kept in touch with Jim. She was paroled after four years. However, marrying again, which was a violation of parole, sent her back to prison to serve the remaining four years. Her chief medical complaint was having "octopuses" under her skin. She did have a gall bladder operation and Jim recalls that she hand picked out the sutures. It healed beautifully when butterflied together.

Another patient was an old-time prostitute who had killed some people. It was known that she had shot one man for running out without payment. She did serve time for attempted murder. However, she always carried a gun, and it was standard practice when she made an office visit for Jim's receptionist to have her check her gun.

In the early years, Fairbanks was known as the "northern terminus of the schizophrenic escape route." Two-thirds of the patients sent to Morningside Hospital in Portland, Oregon from Alaska were from Fairbanks. This was before the days when Alaska had a mental institution and consequently patients were farmed out. There was a favorite saying, "Inside, Outside and Morningside." The evidence of a high incidence of mental illness was shown in the high rate of alcoholism and suicide.

Alice and Jim Lundquist have had eight children. One died of leukemia in 1966. One was killed by a drunk in 1979. Five have stayed in Alaska; one is Outside.

Jim Lundquist, with his excellent educational background, his manual dexterity plus a large amount of steady nerves, and good judgment, is a perfect example of the physicians who came to Alaska at a time when being self-reliant was of prime importance. He has every right to be proud of what he has accomplished. He enjoys the experiences offered to him which were strictly a thing of that time. That medicine has changed there is no argument. But it is exciting to know of one physician who could perform successfully under challenging circumstances. Given all of this, it is to be hoped that Jim Lundquist will someday write his own story.

Gwynneth Gminder Wilson
Chairman, ASMA Auxiliary History Project

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Heart Disease and Diabetes: Deadly Link Uncovered

by William C. Duckworth, M.D.[1]

Heart disease, as most people know, is the leading cause of death by disease in the United States. It is expected to kill some 540,000 people this year.

But here's another fact — one which all too few people know: Diabetes, together with its complications, is the *third* leading cause of death by disease in this country. With its complications, it will cause the deaths of 300,000 Americans in 1987.

New evidence shows a strong and deadly link between diabetes and heart disease. In fact, atherosclerosis (clogging of the arteries, which can lead to coronary heart disease) is the single most common cause of death in adults with diabetes in the U.S.

- Compared with nondiabetic people, approximately twice as many people with diabetes *have heart conditions.* This ratio comes out even higher when women alone are considered.
- Coronary heart disease *is present* in about 13 percent of diabetic adults and congestive heart failure in about 7 percent.
- Heart disease *is involved* in about 50 to 60 percent of all recorded *deaths* of diabetic adults in about 15 percent of fatalities among diabetic children.
- The *risk of death* from heart disease in people with diabetes is about twice that among nondiabetic individuals.
- Heart disease due to blocked coronary arteries is the *direct cause* of at least one-third of all deaths occurring in diabetic patients over 40 years of age.
- Diabetic patients who smoke have an even greater risk of heart disease than do nondiabetics who smoke.

BLOOD FATS PLAY ROLE

According to the American Diabetes Association (ADA), most of the risk factors for atherosclerosis and coronary heart disease are much more prevalent in patients with diabetes than in the general population.

Among the most widely studied of these risk factors are alterations in cholesterol brought about by the faulty metabolism that characterizes diabetes. (Insulin deficiencies or flaws in its action inhibit the diabetic body's ability to turn sugar into energy.)

In untreated non-insulin-dependent diabetes, which accounts for about 80 to 90 percent of all diabetes in the U.S., two prominent components of cholesterol may be altered. The proportion of what is called very low density lipoprotein (VLDL) may increase, and the proportion of high density lipoprotein (HDL) may decrease.

High levels of VLDL are suspected of contributing to atherosclerotic heart disease in some individuals. High levels of HDL, on the other hand, appear to *lessen* the risk of heart disease. Small wonder, then, that the diabetic patient who has developed high levels of VLDL and low levels of HDL is at increased risk for heart disease and, of course, for death from heart disease.

STEPS THAT CAN BE TAKEN

The picture is undeniably grim, but not hopeless. Whereas there is no way known to prevent or cure diabetes, ways *are* known to control the risk factors for heart disease—and that is just what the American Diabetes Association is recommending.

Diet and exercise have long been the cornerstones of non-insulin-dependent diabetes therapy. The objective has been to lower the abnormal blood glucose (sugar) levels that result when the body is unable to convert sugar into energy.

The basic diet for people with diabetes calls for a reduction in total fat and an increased proportion of polyunsaturates. It is virtually the same as the diet recommended by the American Heart Association (AHA) for lowering the high cholesterol levels that so often foreshadow impending cardiovascular disease. Of particular importance to the person with diabetes is that the fat-modified diet reduces VLDL concentrations and often can result in increased HDL levels.

Thus, strict compliance with the diet recommended by the ADA can, in many cases, not only bring down the high blood glucose levels that plague diabetic patients, but also forestall the atherosclerotic buildup that can lead to heart disease.

Although both the ADA and AHA emphasize that diets should be developed to serve each individual's specific needs, both recommend diets that restrict total fat intake to less than 30 percent of total calories consumed. Of that 30 percent, less than 10 percent should be saturated fats (such as in dairy products or meat); unsaturated fats (such as vegetable oil) should make up the other 20 percent. (The average American diet is about 40 percent fat.)

The ADA also notes that current evidence suggests

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[1] Professor of Medicine, Indiana University School of Medicine, Indianapolis, Indiana.

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high fiber diets and soluble-fiber supplements are helpful in improving metabolism, lowering total cholesterol levels and decreasing VLDL concentrations. It says an intake of 40 grams of fiber a day (the average daily intake for most adult Americans is around 10 to 30 grams) can be particularly helpful. And more is acceptable for individuals on weight-reducing diets — a matter of special concern in non-insulin-dependent diabetes, which so often is characterized by obesity.

Exercise is an integral part of any weight-reducing program, of course, but the ADA emphasizes that exercise alone, without concurrent caloric restriction, rarely results in significant weight loss. Still, the ADA says, even light exercise can be quite important in controlling both blood glucose and cholesterol levels.

If diet and exercise fail to cut the cholesterol levels to at least 200 milligrams per deciliter of blood, the physician can prescribe cholesterol-lowering agents.

Robert Levy, M.D., the senior associate vice president for health sciences at Columbia University in New York, quotes one projection that says we could lower the heart disease death toll by 100,000 deaths a year if we could lower the average cholesterol level by just 10 percent.

Effective means of cutting the heart disease toll in the diabetic population, as well as in the population as a whole, exist.

It's a goal worth setting our sights on.

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As a society, we can't let that happen. We must close the gaps in our country's health care insurance coverage so that *everyone* has access to health care—whether that care is given to restore health or to prepare for death. Isn't that our responsibility?

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THE COMMITMENT CONTINUES

President's Page

What a difference a few days of sunshine makes in peoples' everyday attitudes. We are actually seeing more smiles and friendly attitudes. People seem a lot more happy since we have had ten days in a row of sunshine. It makes one forget those cool, cloudy, windy, grey days in June and early July.

Notices will be going out in the near future to both members and non members of ASMA. I encourage you to join or rejoin as the case may be. Whether you agree with all of the policy statements or not, you have to admit that the Alaska State Medical Association is the political voice for organized medicine in this state. ASMA needs your support, not just your dues, but your time and efforts in various endeavors in the coming year.

The number one effort should be that of continued work toward obtaining effective Tort Reform. Efforts now of the ASMA are being funneled into getting an initiative on the ballot in the fall of 1988 regarding Tort Reform. This is going to take a lot of time, a lot of effort, and a lot of money. We need your support. ASMA also has various and sundry services to offer you as a member. These include transcription services, collection services, our monthly newsletter "Heartbeat," and the ever increasingly sophisticated journal *Alaska Medicine*. There are a number of standing committees of ASMA in which your services are needed. If you have a special interest in any area and want to become active in a committee, please call the staff at ASMA or the committee chairperson.

We also hardily endorse membership in the American Medical Association. The AMA is a very strong, very well-organized society, and it has the strongest voice for organized medicine nationwide. For the same reasons that we need you in ASMA, your membership is also desired on the national level in the AMA.

Our next annual meeting will be in June 1988 in Fairbanks. Staff of ASMA are already gearing up to have an outstanding convention. Dr. Rothrock of Fairbanks is the Convention Committee chairman, and Dr. Charles Steiner of Fairbanks is the CME Committee chairman. They will be responsible for the clinical hours to be presented at the convention. If you have any desire to help on these committees, please contact those chairmen.

Hope you have a crisp and enjoyable fall season.

Jerry Little, M.D.
President
Alaska State Medical Association

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Executive Privilege

Efforts to obtain meaningful tort reform in Alaska continue in spite of our setback in the 1987 legislative session. We were successful in obtaining 11 cosponsors out of 20 members in the Senate but were unable to move the bill out of the Judiciary Committee. Because of political pressure, some of our staunchest supporters in the past have been less than supportive recently in their efforts to move the bill out of Committee. Because of this recalcitrance on the part of the Legislature to do anything further with tort reform, we have elected to pursue the initiative option. To that end, the Citizens Coalition for Tort Reform which represents over 40 groups and the Health Care Coalition which represents the major health provider groups in the state have agreed to work closely together on the initiative.

At this date the initiative is currently on the Lieutenant Governor's desk for his approval. He has 60 days from the time of submission to approve or reject the initiative. We are in hopes that he will approve it expeditiously so that we may begin gathering the 18,256 signatures that are necessary to put this on the ballot. Needless to say, we will want to gather a great deal more signatures than required. We are hopeful that each of you will help us in this effort by becoming a sponsor.

There are two basic issues addressed by the initiative. The first part of the initiative would remove joint and several liability from Alaska law in its entirety. Put simply, this would mean that everyone who was a defendant would be held responsible for only their portion of the fault. Another way of stating this is that if your fault is determined by the judge or jury to be 10 percent, you pay 10 percent of the total liability, and not 20 percent as present law would now have it. Similarly, if you are 60 percent responsible, you pay 60 percent and not the entire bill as the present law now states. The trial lawyers oppose this provision because by naming a deep pocket such as a hospital, an insured doctor, a municipality, etc., they insure themselves of being paid entirely if they do win a verdict. Pure several liability would say simply that everyone is responsible for their actions, but they are not responsible for anyone else's actions.

The second part of the initiative would limit the contingent fee that may be claimed by an attorney in a personal injury case to 33 1/3 percent of the first $100,000, 25 percent of the second $100,000, and 10 percent of any amount over $200,000. This is a significant departure from the present situation, which allows as much as 50 percent of the entire amount plus expenses.

We believe that both of these provisions are effective tort reform measures. Furthermore, they are very simple and easy to explain to persons who are not skilled or versed in tort law. Finally, they are fair, and we believe they are well supported by the populace at large. A recent poll shows that in the neighborhood of 75 percent of the voting members of the State of Alaska approve of tort reform.

There have been endless discussions about the need to obtain other tort reform measures. No one would argue that this initiative is not as complete as we might hope it to be. Nevertheless, it represents two significant issues. These issues are at the same time relatively simple and easy to explain, and therefore we believe we can capture a victory should we be on the ballot in 1988. We hope to gather sufficient signatures so that the legislature is persuaded of the likelihood of passage of this initiative, and therefore will pass it into law in the legislative session of 1988. The legislature will have the 1988 session to address this, or else the measure will appear on the ballot.

This will be a concerted and difficult effort and will need help from every quarter in order to amass the requisite number of signatures. But, your association is resolved to accomplish this, with your help.

Raymond Schalow
Executive Director
Alaska State Medical Association

Auxiliary News

Alaska State Medical Association Auxiliary

In response to the non-response of some of our state legislators as regards tort reform, the Citizens Coalition for Tort Reform (CCTR) is aiding an initiative to appear on the ballot in the fall of 1988. The initiative addresses the joint and several liability problem, and, if adopted, will finally find fault where it is due and not just attack the deep pocket.

The initiative also calls for a sliding scale contingency fee for attorneys. Payment would be more fair and just.

Help is needed from the auxiliary. If you can work as little as several hours on one weekend, please call me. The donation of some of your time will help make all our lives less stressful.

Cassie Hill
President, ASMA Auxiliary

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Guest Editorial

An Alice-in-Wonderland attitude about the financing of medical and health services is pervasive in this country.

Significant expansions of health care programming are enacted or proposed, but little attention is paid to where the money to pay for it will come from. Various social and health programs were enacted in the 1960s with much political and public fanfare. To curry votes and public favor, the eventual costs of programs such as Medicare and Medicaid were drastically underestimated, as the AMA, among others, pointed out at the time.

But when all the bills kept going up and coming due, some of the programs dwindled and some have disappeared. And since present appropriations rarely live up to past political promises, successive, steep budget cuts in projected expenditures for Medicare and Medicaid have been made, with more in the wings. And we have the DRG and PRO programs to control hospital utilization. Prospective pricing programs — a la managed care plans — also have become popular in the private sector.

Meanwhile, spendthrift hospitals and physicians have somehow become the villains of the piece.

This, in spite of the fact average life expectancy in this country is now almost 75 years, up from 47 years at the turn of the century. Today there are more Americans aged 65 and older than the entire population of Canada. Between 1970 and 1984, the number of people aged 85 and over jumped 90%, from 1.4 million to 2.7 million. And the AMA predicts that by the turn of the next century, the number of Americans over 75 will more than double and those over 85 more than triple.

Ironically, of course, this is precisely why programs such as Medicare have gotten into deep financial trouble. As currently structured, it is estimated that part A of the trust fund will be bankrupt by the year 2002. By the year 2017 it will be in debt one trillion dollars. And by the year 2010, U.S. entitlement programs will consume two-thirds of the federal budget.

As physicians, we have an obligation to make patient care as cost-effective as possible. Our profession —including our federation of organized medicine — is engaged in a number of initiatives to help meet that need.

But we must never let bottom lines and balance sheets interfere with our overriding commitment to stand as first advocates for our patients, and the quality and availability of care they need, as well as its adequate financing. Just last year the AMA publicly demonstrated that commitment by proposing massive restructuring of the Medicare system, which would convert it to a sound actuarial basis.

And we can count on public support. Our annual public opinion surveys consistently show that while the total cost of care is perceived as a problem, a majority of the American people believe we're not spending enough on health care. In our 1986 poll, 54% of the respondents said this was true, and 70% said more should be spent on health care programs for the elderly. Medicare means testing was favored by 60%, and 70% said they would be willing to pay more taxes to help resolve Medicare financing problems.

In addition to added health and life, another reason for that support is something legislators rarely if ever mention — *human* capital. Theodore Schultz, the noted economist, has described it this way: "Clearly, the value productivity of human beings in the United States is now vastly larger than all other forms of capital taken together."

Millions of Americans are now alive who would not have previously survived. And they're leading healthful and productive lives, earning and spending billions of dollars, adding to the common economic and social good. When they do have health problems, most are quickly treated and returned to the workplace, saving business and industry additional billions of dollars in what would otherwise have been lost workdays and productivity.

Benjamin Disraeli, the 19th Century English statesman, had a more elegant description. He said, "The health of the people is really the foundation upon which all their happiness and all their powers as a state depend."

What our own nation, *as* a nation, has built is a high and beneficial plateau of better health and a better quality of life for the American people. And in the last analysis, however patient care is financed or in what forms it's delivered, I believe this nation, *as* a nation, will continue to believe that nothing is more important than that.

William S. Hotchkiss
President
American Medical Association

Editorial

The Alaska State Medical Association has provided significantly increased support for *Alaska Medicine*. The journal is an important scientific forum to report important research findings and to facilitate scientific and professional examination of major issues affecting medicine, public health, and health care services delivery. But the reality of science is that scientific journals, however prized, are rarely economically self-sustaining. *Alaska Medicine* is no exception. Humana Hospital provided *Alaska Medicine* with a major grant to underwrite the entire cost of publication of this issue. Humana's support is greatly appreciated. Our journal continues to grow and its potential is bright. Publication of the journal can enhance the health of generations of present and future Alaskans — and be the place where you can read the facts.

This past weekend, a friend told me he used to begin his first lecture to his college English class by saying, "the facts never speak for themselves." The truth of his comment was brought home to me recently in a most telling incident. It reminded me also that our memories are short.

This past week a major Alaska newspaper article reported on efforts of parents, members of an organization called Dissatisfied Parents Together (DPT), to change Alaska's immunization law by weakening current requirements for childhood vaccination. Little recognition was given to the serious consequences of these diseases and none to the tremendously successful program that has been in existence since March 1, 1977. On that day, 2,300 Alaskan school children were excluded from school because they were not adequately vaccinated. All but 52 were back in the classroom, properly vaccinated, within two weeks. Since 1977, the 95,000 school children in Alaska's classrooms each year have met immunization requirements. The program has been an outstanding success. Although people have brought measles, rubella, and pertussis into Alaska, since 1977 no Alaskan school children have contracted measles, rubella, pertussis, polio, diphtheria, or tetanus. Spread of disease has not occurred because immunizations levels have been so high. We need only to remember the past — we can then see clearly that our program is essential and must not be changed. We must provide the facts. Once they are remembered, the benefits of the program will be evident.

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BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate.

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Carcinogenesis, Mutagenesis, Impairment of Fertility: No evidence of drug-related tumorigenicity was found in chronic oral toxicity studies of 24 months' duration conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies have not been conducted.

Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

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DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

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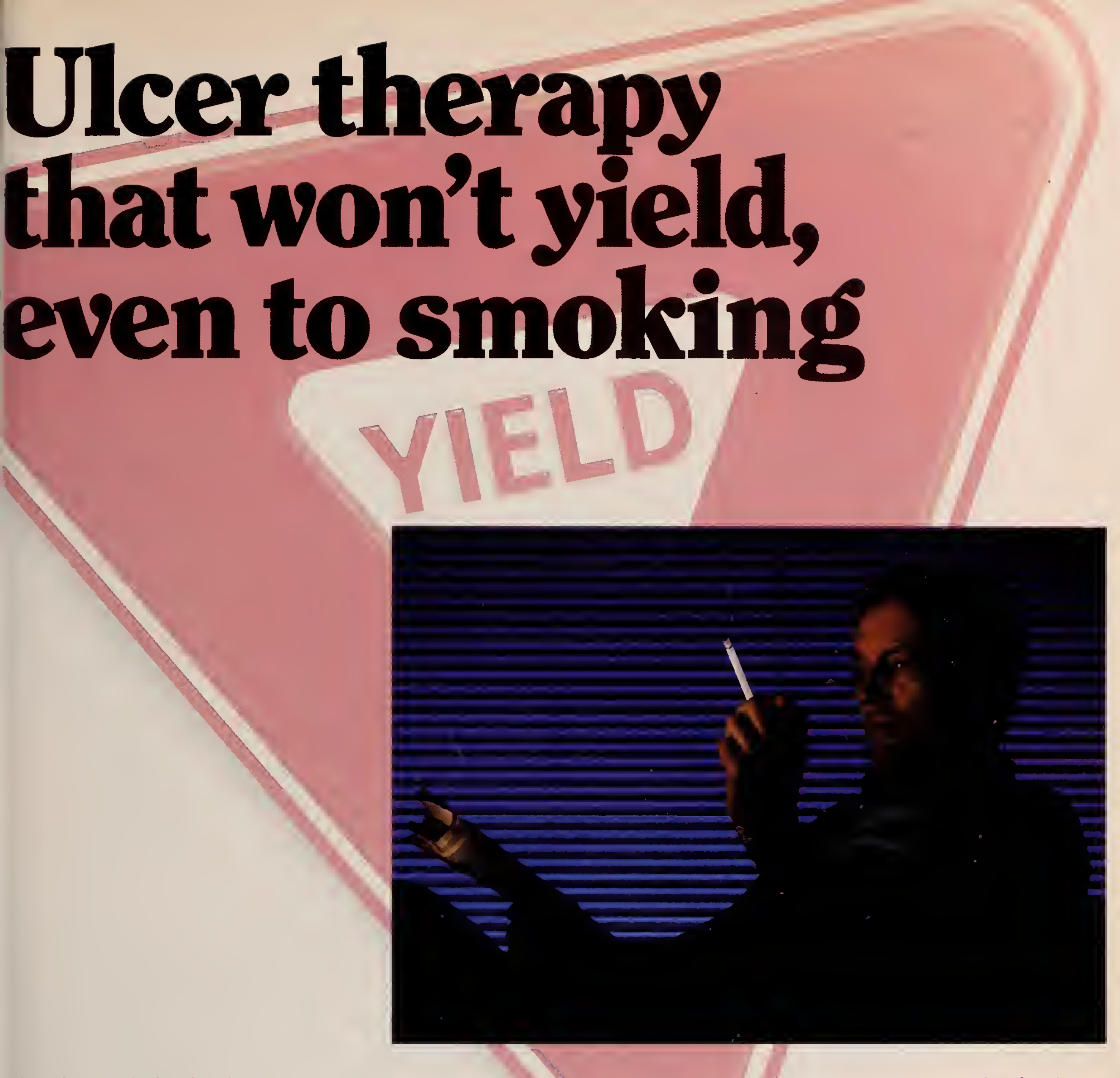

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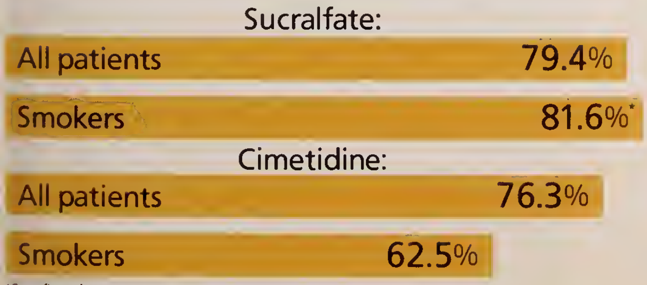

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PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

1 ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA
Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year This risk was independent of the other known risk factors for endometrial cancer These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13 9 times greater than in nonusers The risk appears to depend on both duration of treatment and on estrogen dose In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration, it therefore appears prudent to utilize such a regimen Close clinical surveillance of all women taking estrogens is important In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
2 ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects One case-controlled study estimated a 4 7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion) Some of these exposures were very short and involved only a few days of treatment The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters Tablets are available in 0 3 mg, 0 625 mg, 0 9 mg, 1.25 mg, and 2 5 mg strengths of conjugated estrogens Cream is available as 0 625 mg conjugated estrogens per gram
INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP)· Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions) Osteoporosis (abnormally low bone mass) Atrophic vaginitis Kraurosis vulvae Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING)
Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS) The choice of progestin and dosage may be important, product labeling should be reviewed to minimize possible adverse effects
CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions. 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (see Boxed Warning). 4 Undiagnosed abnormal genital bleeding 5 Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy)
WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning) However, a recent large, case-controlled study indicated no increase in risk of breast cancer in postmenopausal women. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrogens per day) comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tende abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estr containing oral contraceptives Increased blood pressure may occur with use of estrogens in the menopau blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been obser patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be ca observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metasta
PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic o and should include a Papanicolaou smear As a general rule, estrogen should not be prescribed for longe one year without another physical examination being performed Conditions influenced by fluid retention as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation Certain patien develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine ble mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase t of endometrial hyperplasia in some patients Oral contraceptives appear to be associated with an inc incidence of mental depression Patients with a history of depression should be carefully observed Pre-ex uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of es therapy when relevant specimens are submitted If jaundice develops in any patient receiving estroge medication should be discontinued while the cause is investigated Estrogens should be used with c patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercal or in young patients in whom bone growth is not yet complete If concomitant progestin therapy is used, po risks may include adverse effects on carbohydrate and lipid metabolism

The following changes may be expected with larger doses of estrogen·

a Increased sulfobromophthalein retention

b Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinep induced platelet aggregability

c Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormo measured by PBI, T_4 by column, or T_4 by radioimmunoassay Free T_3 resin uptake is decreased, reflecti elevated TBG, free T_4 concentration is unaltered

d Impaired glucose tolerance

e Decreased pregnanediol excretion.

f Reduced response to metyrapone test

g Reduced serum folate concentration.

h Increased serum triglyceride and phospholipid concentration

As a general principle, the administration of any drug to nursing mothers should be done only when necessary since many drugs are excreted in human milk

Long-term, continuous administration of natural and synthetic estrogens in certain animal species inc the frequency of carcinomas of the breast, cervix, vagina, and liver However, in a recent, large case-con study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estr
ADVERSE REACTIONS: The following have been reported with estrogenic therapy, including ora traceptives. breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea, premenstru syndrome, amenorrhea during and after treatment, increase in size of uterine fibromyomata, vaginal candi change in cervical erosion and in degree of cervical secretion, cystitis-like syndrome, tenderness, enlarg secretion (of breasts), nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice, chloas melasma which may persist when drug is discontinued, erythema multiforme, erythema nodosum, hemo eruption, loss of scalp hair; hirsutism, steepening of corneal curvature; intolerance to contact lenses, hea migraine, dizziness, mental depression, chorea, increase or decrease in weight, reduced carbohydrate tol aggravation of porphyria, edema, changes in libido
ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females
DOSAGE AND ADMINISTRATION:
PREMARIN® Brand of conjugated estrogens tablets, USP
1 *Given cyclically for short-term use only* For treatment of moderate-to-severe *vasomotor* symptoms, a vaginitis, or kraurosis vulvae associated with the menopause (0 3 mg to 1 25 mg or more daily) The lowes that will control symptoms should be chosen and medication should be discontinued as promptly as po Administration should be cyclic (eg, three weeks on and one week off) Attempts to discontinue o medication should be made at three- to six-month intervals
2 *Given cyclically* Osteoporosis Female castration Osteoporosis —0 625 mg daily Administration sh cyclic (eg, three weeks on and one week off). Female castration—1 25 mg daily, cyclically Adjust up downward according to response of the patient. For maintenance, adjust dosage to lowest level that will effective control

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate me taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding
PREMARIN® Brand of conjugated estrogens Vaginal Cream
Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae

The lowest dose that will control symptoms should be chosen and medication should be discontin promptly as possible

Administration should be cyclic (eg, three weeks on and one week off)

Attempts to discontinue or taper medication should be made at three- to six-month intervals

Usual dosage range· 2 g to 4 g daily, intravaginally, depending on the severity of the condition

Treated patients with an intact uterus should be monitored closely for signs of endometrial canc appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or re abnormal vaginal bleeding

References:

1. Lindsay R, Hart OM, Clark OM The minimum effective dose of estrogen for prevention of postmenc bone loss *Obstet Gynecol* 1984,63.759-763. **2.** Studd JWW, Thom MH, Paterson MEL, et al: The prevent treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pas Paoletti R, Ambrus JL (eds). *The Menopause and Postmenopause.* Lancaster, England, MTP Press Ltd chap 13

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Letters to the Editor

Joe Shelton on Merrill Pass

Dear Editor:

One of the first Alaska docs I met on arrival in Alaska 20+ years ago was Dr. Joe Shelton. Joe retired from the practice of ophthalmology a few years ago and is now in Salt Lake City in very fragile health. Joe is a real gentleman and I feel privileged to have known him.

Joe was also a very good bush pilot. After I obtained an airplane and started exploring Alaska, Joe took me aside one day and briefed me on getting through Merrill Pass without adding to the pile of bent tin that decorates that particularly awesome piece of real estate. I am certain that he briefed others similarly, but since numerous aviating docs have arrived in recent years, I will pass along his pointers. Obviously, these become important only when weather conditions are marginal and one is forced to fly low. One should not make one's first transit of the pass in these conditions—but rather should go through several times on a nice day at progressively lower altitudes—it looks much different from a few feet above the deck than from a few hundred feet, and at minimum altitude it is too narrow to turn around.

When the pass is approached westbound, one should stay on the right side of the valley after making the right turn above Kinibuna Lake. As the end of the valley gets uncomfortably close, the pass is to the left. A 180 degree turn is made to the left while looking through the pass. If the cliffs beyond it are clearly visible, a 270 degree turn is made and the pass entered. One should stay on the left side as the pass is exited so that the long valley leading down to TwoLakes can be foresighted. If that valley is full of weather, there is room for a steep 180 degree turn to re-enter the pass and head for more friendly terrain.

Eastbound, one flies up the valley from TwoLakes staying on the right side. As a 180 degree turn is started, one can see through the pass. If it is blocked by weather, the turn can be completed to retreat. If it is open, the turn is converted to a 90 degree, the pass transited staying on the left side so that one can look down toward Kinibuna Lake. If the valley is plugged, a 180 degree turn can be made over the wreckage of the C-47 and the pass reentered.

Those who die in Merrill Pass fail to make the turns necessary to insure that the pass is open, fly directly into it, encounter a wall of fog, and add to the litter. I can't say that Joe's briefing has saved my life, but certainly it has allowed me to complete several trips that I might not have attempted, and my heart rate has remained closer to normal—a consideration that increases in importance with age and degree of coronary atherosclerosis.

Today there are at least five wrecked airplanes clearly visible in the pass. Others have been ground away in the rocks. Perhaps Joe's clues will help some of you avoid the trap.

I think it would be fitting if the abrupt peak that forms the south side of the pass could be named for Joe. Mt. Shelton has a nice ring. I have obtained the necessary paperwork to attempt this. If anyone agrees, let me have your thoughts.

Donald R. Rogers, M.D.
Pathologist
2801 Debarr Road
Anchorage, Alaska 99508

Annual Pathology Meeting

The Annual Meeting of the United States and Canadian Academy of Pathology will be held at the Washington Hilton in Washington, DC starting with "Companion Meeting" symposia on Sunday, February 28 and continuing through Friday afternoon, March 4, 1988.

Scientific Papers, Poster Sessions, thirteen Specialty Conferences, and 49 Short Courses are scheduled. Two Special Courses will be offered on "Immunopathologic Techniques in Diagnostic Pathology" with Drs. Robert McCluskey and Robert Colvin as Course Directors and "Electron Microscopy in Surgical Pathology" with Dr. Bruce MacKay as Course Director. The Long Course will be on "Gastrointestinal Pathology" with Drs. Harvey Goldman and Henry Appelman as Course Directors.

The Maude Abbott Lecture will be delivered by Dr. Marilyn Farquhar on Tuesday, March 1.

Timely Topics in Pathology will feature a session on "Technology Transfer - The New Molecular Pathology, Opportunities for the Future" presented by Dr. Cecilia Fenoglio-Preiser.

Further information may be obtained from Dr. Nathan Kaufman, Secretary-Treasurer, United States and Canadian Academy of Pathology, Inc., Building C, Suite B, 3515 Wheeler Road, Augusta, GA 30909. Telephone (404) 733-7550.

Calendar of Events

EMS Symposium
Anchorage, Alaska
November 12–14, 1987

The 12th Annual Alaska Symposium on Emergency Medical Services will offer workshops on emergency care, injury prevention, public information, and management of emergency services, with particular emphasis on the recognition of the volunteer EMS provider's contributions to the care of the sick and injured in Alaska.

These workshops will provide continuing medical education credits applicable for Emergency Medical Technician and Mobile Intensive Care Paramedic recertification requirements, CEARP credits for Nurses, and Category I continuing medical education requirements for physicians and physicians assistants. Contact: Charles Ramage, Associate Coordinator, Emergency Medical Services Section, Division of Public Health, Department of Health and Social Services, Box H-06C, Juneau, AK 99811-0616, (907) 465-3027.

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Caution patients about the combined effects of Limbitrol with alcohol or other CNS depressants and about activities requiring complete mental alertness, such as operating machinery or driving a car. In general, limit dosage to the lowest effective amount in elderly patients.

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References: 1. Feighner JP, *et al: Psychopharmacology 61* 217-225, Mar 22, 1979 **2.** Data on file, Hoffmann-La Roche Inc, Nutley, NJ

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Tranquilizer—Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use, then initiate cautiously, gradually increasing dosage until optimal response is achieved Contraindicated during acute recovery phase following myocardial infarction
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs Closely supervise cardiovascular patients (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e g , operating machinery, driving)

> ***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage, withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide)
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients Periodic liver function tests and blood counts are recommended during prolonged treatment Amitriptyline component may block action of guanethidine or similar antihypertensives When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady state concentrations of the tricyclic drugs Concomitant use of Limbitrol with other psychotropic drugs has not been evaluated, sedative effects may be additive Discontinue several days before surgery Limit concomitant administration of ECT to essential treatment See Warnings for precautions about pregnancy Limbitrol should not be taken during the nursing period Not recommended in children under 12 In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects
Adverse Reactions: Most frequently reported are those associated with either component alone drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs
Cardiovascular Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke
Psychiatric. Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido
Neurologic Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns
Anticholinergic Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract
Allergic Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus
Hematologic. Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia
Gastrointestinal. Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue
Endocrine Testicular swelling and gynecomastia in the male, breast enlargement galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion
Other Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling
Overdosage: Immediately hospitalize patient suspected of having taken an overdose Treatment is symptomatic and supportive I V administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning See complete product information for manifestation and treatment
Dosage: Individualize according to symptom severity and patient response Reduce to smallest effective dosage when satisfactory response is obtained Larger portion of daily dose may be taken at bedtime Single *h.s* dose may suffice for some patients Lower dosages are recommended for the elderly Limbitrol DS (double strength) Tablets, initial dosage of three or four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required Limbitrol Tablets, initial dosage of three or four tablets daily in divided doses, for patients who do not tolerate higher doses
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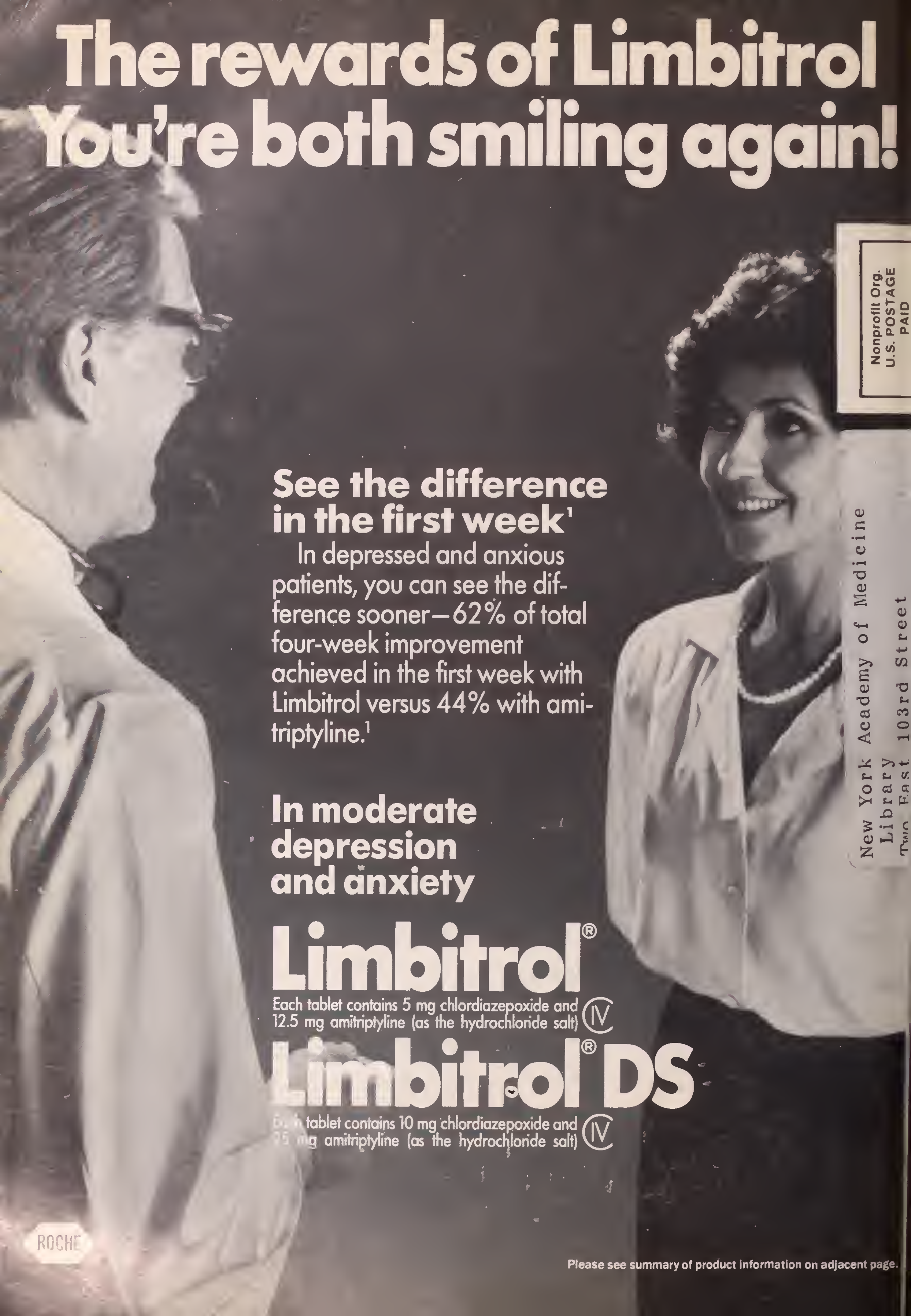

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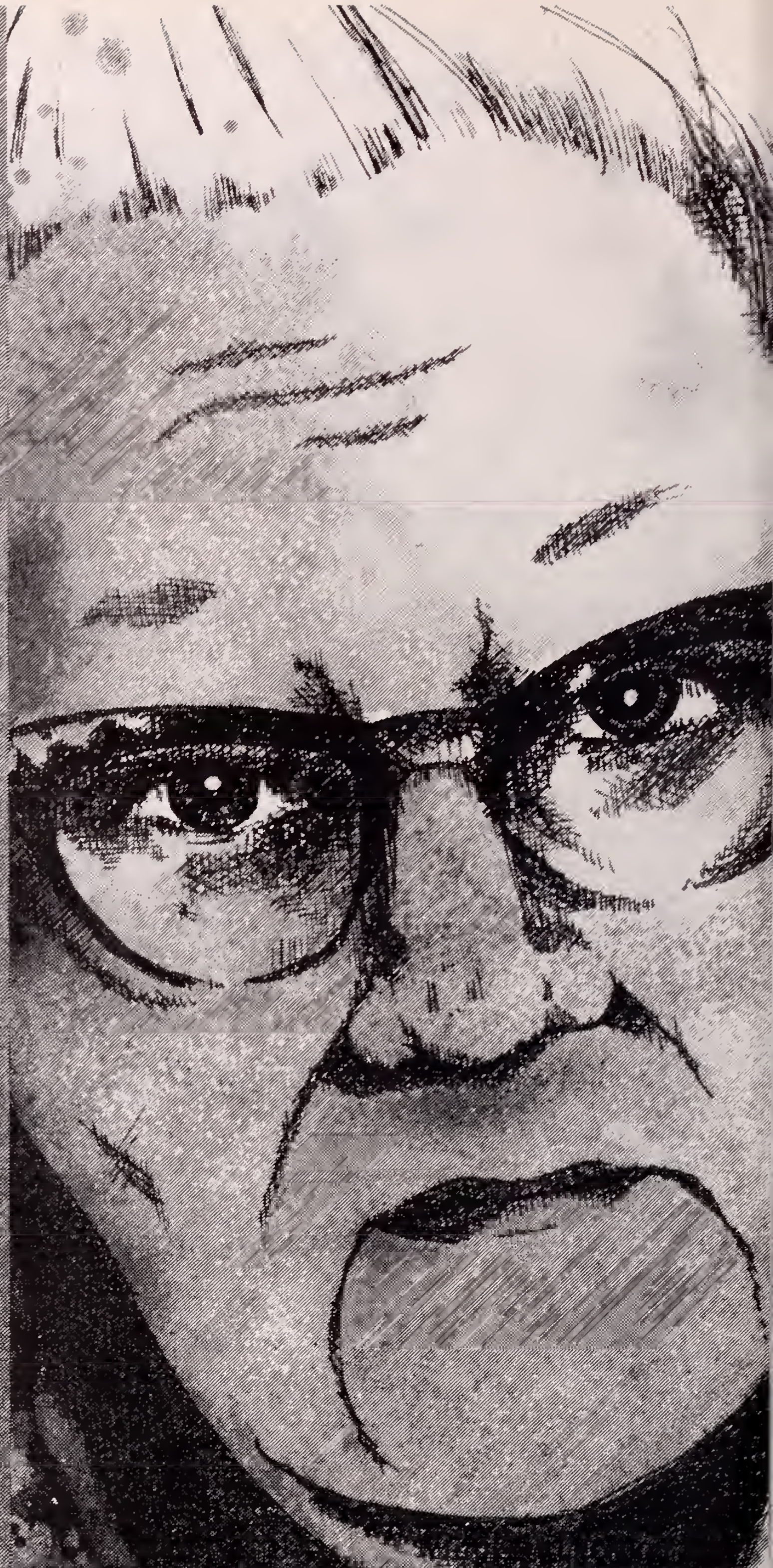

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THE COMMITMENT CONTINUES

Nicaraguan Health Care and the Contra-War

Robert A. Mack[1]

ABSTRACT

Nicaragua has made impressive strides in health care provision since 1979. These include a significant reduction in the infant mortality rate and in a number of critical childhood infectious diseases. Much of this progress has come from the reorganization of the health care system which now focuses more on primary health care and appears more in balance with the country's rural needs. The Contra War, however, now seriously threatens these achievements in two basic ways: (1) through the direct contra policy of kidnapping, torture, intimidation, and death of health personnel and destruction of health facilities, and (2) though less direct, the general destabilization of the economy through strategic destruction of coffee crops, storage facilities, selective community intimidation, and U.S. embargoes and loan blockades, which all conspire to mitigate many important advances already made in Nicaragua's health care system.

INTRODUCTION

The Nicaraguan government has made impressive strides in health care during the past 5-6 years, earning the World Health Organization's annual award in 1982 for the most significant achievement in public health.

These gains, however, are seriously threatened by the expanding U.S. supported Contra War.

HEALTH CARE GAINS

Some of the advances include:

1. a significant drop in the rate of infant deaths from one of the highest in Central America to one of the lowest (120-140/1000 live births to 79/1000 live births) (1-3),
2. development of a national system of immunization that has dramatically reduced or eliminated several important childhood diseases like polio, diphtheria, measles, and tetanus. Effective anti-malarial campaigns have also been conducted, and
3. successful initiation of preventive health care, illustrated by the government's construction and distribution of some 4,200 latrines, numerous clean water projects, house construction, and an evolving nutritional program that reaches over 100,000 children per year. The latter has been instrumental in reversing a previous 10 year statistic in which Nicaragua recorded a 105% increase in child malnutrition, the second highest in Central America (2).

Much of this progress has been the product of reorganization of the health system that accompanied the revolution. Formerly, over half of all hospital beds were concentrated in three major cities, and only five health facilities with beds were in the rural area, mostly staffed with untrained auxiliary nurses. Health professionals, as well as potable water and sewage systems, followed a similar skewed distribution (this in a country over 50% rural population) (1,3). The increased construction of rural hospitals and clinics and the development of mobile health units have dramatically changed health care access for the rural population.

An additional vital part of this restructuring, affecting rural areas particularly, was a 1982 nationwide training program established to train parteras (midwives). By the end of 1984, over 3,000 parteras had been retrained and incorporated into the national health care system. This particular act has had significant effect on neonatal deaths, largely through controlling neonatal tetanus (4).

Of the hundreds of primary care centers built over the past few years, many have successfully served as oral rehydration centers, estimated to have served a notable 92,000 children, most under the age of one year. These reversals in the neglect of the rural poor have been matched by the increased number of health professionals. An additional medical school opened in Managua and the existing one in Leon was expanded, while two new nursing schools opened outside Managua (1,3).

A further unique development has been the opening of an office in Esteli for the rescue of popular medicine. This office, after compiling scientific data on the medical value of various plants, coordinates the collection and promotion of the most useful of them. Importantly, too, the office serves to familiarize practicing physicians with its results through the reestablishment of a "popular medicine clinic" (5).

It is precisely such advances in health care that the current U.S. directed contra war directly threatens.

EFFECTS OF THE CONTRA WAR

The most brutal and direct effect of the Contra War is its uncommon focus on the health care system itself.

In many areas of the country, selected destruction of hospitals, health clinics and pharmacies has deprived an estimated 10% of the Nicaraguans some degree of access to health services. And 1984 showed for the first time

---

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since 1979 an 8% reduction in per capita medical visits and a fall of 6% per capita hospitalizations (6). Also, though tetanus and measles no longer threaten health as they once did, their slight rise in 1984 is an ominous sign of the war's effects. Health care workers of all ranks, including foreign physicians, continue to be threatened—targeted for torture, kidnapped and killed (7–9). The Contras seem intent on removing health care advances particularly in the rural areas and perhaps as Bossert states, "...the vengeance of the attacks can best be seen as a measure of the health care system's success" (1).

The contra war also affects health systems through its direct economic impact and its terroristic nature. Government officials estimate the war has cost Nicaragua $1.3 billion, an amount comparable to 4 years of export earnings (5).

Currently, nearly 60% of the national budget is allocated to the war effort (10); and fueled by having 20% of its most economic active population in the military, inflation soared to 334% in 1985 (11). Moreover, the pressure of the U.S. blockade and its accompanying veto power on international loans conspire to make long range health planning as part of economic development an impossibility (10,12).

An auxiliary nurse from the Nueva Guinea area recently told me, "...the last year has gotten worse for me because it's getting harder and harder to get medicines, antibiotics especially...they tell me there is a shortage and they're trying. I'm told money for medical supplies is short...." (13). A quick glance at the barren supply cabinets confirmed her concerns. She expressed frustration that she won't be able to make better use of newly developed skills and a sadness at the realization that future hopes for improving her medical knowledge were probably nonexistent.

In 1984, Nicaragua spent 10% of its national budget on health. The escalation of the war as represented by the recent U.S. congressional vote endangers such a commitment. Already established successful programs are in clear jeopardy. Recently price increases of 300% on beans and 100% on rice, sugar and milk, all basic foods (11), raise the real threat of nutritionally induced diseases.

Acts of economic destabilization are particularly effective as carried out by the Contras. One example of this involves coffee, a major cash crop. Though last year's crop was successfully harvested and processed, in several past seasons the Contras have sabotaged significant portions of coffee harvests and storage facilities. According to one report, "... in 1985 they paralyzed an estimated 24% of the coffee crop's harvesting, processing and transport" (14). As part of this process of sabotage, the Contras have successfully terrorized local villages through assassination, torture, and kidnapping.

I found a recent example of the effectiveness of this strategy in February and March of this year. In the small village of Los Angeles, in the Nueva Guinea Zone, most of the coffee had dried up and remained unharvested. This, a direct result of the justified fear people felt in going to the fields, stemmed from two recent Contra attacks associated with torture, kidnapping and killing of unarmed village farmers.

As these terroristic acts foster increased general instability, they also lead to large population relocation. Perhaps as many as 200,000 Nicaraguans have become internal refugees, fleeing their homes in war contested areas, and in the process absorbing some $66 million in relief and relocation costs (2,6). In many cases these migrants end up in urban areas where, in crowded settings and stressed resources, they contribute to an increase in infectious disease. The large epidemic of Dengue Fever in Nicaragua during the summer of 1985 has been cited as deriving in part from such processes.

Considering all the foregoing, the prospects for continued effective health care would seem dire were it not for the Nicaraguan determination.

As I watched two pickup truck loads of volunteer health workers–brigadistas leave the town of Jinotega to begin another Health Care Day, I reflected on their megaphone delivered call, "...salud es revolutionario...," and thought yes, effective health care for the majority in the context of third world countries is indeed revolutionary.

END NOTES

Other significant health progress not discussed include areas of dental care, occupational health and safety and toxic waste. Also significant is the successful formation of a number of support groups dealing with psychological trauma and mental health problems related to the war.

Some mention ought also to be made of the dramatic effectiveness of the literacy campaign carried out early in the revolution in which the illiteracy rate of 59% (with some rural areas having 90%) was reduced to about 12–13%. Obviously literacy is a basic necessity in any serious effort to improve health care. Because of the Contra War there are now some suspicions that the illiteracy rate is climbing, understandable when considering that the median age for Nicaragua is 15 years.

Robert Mack teaches Medical Anthropology and Health Issues at the University of Alaska, Anchorage, AK. The article is based on a six-week visit to Nicaragua, February–March 1986 and subsequent library research while on sabbatical in Tucson, AZ, and Anchorage, AK, as well as correspondence with health workers in Nicaragua.

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The Epidemiology of Suicide in Alaska, 1983-1984

by W. Gary Hlady, M.D.[1]
and John P. Middaugh, M.D.[2]

ABSTRACT

According to the National Center for Health Statistics, Alaska has one of the highest rates of suicide in the nation. This study describes the epidemiology of suicide in Alaska on a statewide basis using data obtained from a manual review of death certificates, supplemented and validated by information in autopsy records. During 1983-1984, 195 deaths were legally determined to be suicides as indicated on the death certificates. The ratio of male-to-female suicides was 4.9 to 1. Suicides occurred more than twice as often and at younger ages among Natives than among Whites. The majority of deaths resulted from gunshot wounds in all race/sex categories, and suicide by firearms was associated with acute alcohol intoxication ($p<.001$, odds ratio 1.3). The average annual rate of years of potential life lost to suicide before age 65 for Natives was 1,684 per 100,000 population, 3.4 times the rate for Whites. These results clearly define young Native Alaskan males as a very high-risk population and suggest that preventive efforts directed at this group may be more effective than crisis intervention.

INTRODUCTION

According to previously published data, the suicide rate for 15- to 24-year old Alaskans increased 277% from 1964 to 1987 (1). Among Native Alaskans, suicide ranked second only to accidental injury as a cause of death during the years 1974-1976 (2). In 1980, Alaskan Natives represented 26% of the State's suicides although they accounted for only 16% of the population (3).

These summary statistics were derived from vital statistics records maintained by the National Center for Health Statistics (NCHS) and the Alaska State Bureau of Vital Statistics. We have recently shown that these data sources underrecorded suicides in Alaska by at least 43% and 28%, respectively, for 1983 and 1984 (4).

An accurate description of the problem of suicide is essential to plan and evaluate programs aimed at prevention. This study describes the epidemiology of suicides in Alaska on a statewide basis, using data obtained from a manual review of death certificates, supplemented and validated by information in autopsy records.

METHODS

We defined suicide as any death occurring in Alaska during calendar years 1983-1984 that was determined by local reporting officials to be suicide, as indicated on the death certificate by listing suicide as either the underlying cause or the manner of death.

The State of Alaska was divided into 38 recording districts. Two districts, Anchorage and Fairbanks, were staffed by professional coroners. The remainder were staffed by recording magistrates who often also functioned as sheriff, justice of the peace, etc.. All cases of suspected suicide were required to be investigated. The usual process was for the local official to collect evidence and testimony in cooperation with police and the medical examiner. A coroner's inquest was then held before a six-person jury to determine whether the manner of death was suicide, homicide, accidental, natural, or undetermined. The inquest was sometimes waived when the manner of death was clearly apparent and uncontested.

Determination of suicide rested upon evidence of intent, but there were no standard criteria by which to evaluate the evidence. The final determination was entered on the death certificate. One copy of the death certificate remained in the local files and the original was sent to the Bureau of Vital Statistics in Juneau. Copies of death certificates were sent from Juneau to NCHS for International Classification of Disease (ICD) coding of the underlying cause and manner of death. These ICD codes were entered along with other information on the death certificates into the state's computer files, from which all official reports were made.

To identify cases, in January 1986 we manually searched all death certificates on file in the Alaska State Bureau of Vital Statistics for the years 1983 and 1984. Reports of all autopsies performed on suicide victims were also reviewed.

Annual midyear (July 1) population estimates were obtained from the Alaska Department of Labor, which strictly follows U.S. Census Bureau definitions and standard demographic methodologies (5). Population estimates for January 1, 1984, were an average of the 1983 and 1984 mid-year estimates. Average annual age-adjusted rates were calculated by the direct method, using the 1940 U.S. population as standard so results would be comparable to figures published by the Centers for Disease Control (CDC) (6). Statistical analyses were performed on pooled data from the 2-year period using the Statistical Analysis System (SAS). Years of potential life lost (YPLL) were calculated for suicide victims less than 65 years of age by subtracting the age at death (in years) from 65.

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RESULTS

Distribution by Sex, Race, and Age

One hundred ninety-five suicides met the case definition of suicide (Table 1). Although males represented 53.4% of the population, they accounted for 83.1% of the suicides. The ratio of male-to-female suicides was 4.9 to 1. The median age for male suicides was 28 years. The median age for female suicides was 25 years.

Two suicides were in Blacks, 128 were in Whites, and 65 were in Natives (Figure 1). Natives accounted for 14% of the population and 33% of suicides.

The median age for all suicides was 28 years. The median age of 23 years for Native suicides was nine years younger than the median age of 32 years for White suicides.

Rates

Crude suicide rates by race and sex are shown in Table 2. The average annual age-, race-, and sex-specific suicides rates (Figure 2) were calculated from some very small numbers and must be interpreted with caution. For example, the rate of 257 per 100,000 Native males in the 20- to 24-year age group was based on 20 suicides over two years in a population of 3,888.

The average annual age-adjusted suicide rate was 21.0 per 100,000 population. Among Natives it was 42.9 per 100,000, 2.2 times the rate of 19.1 for Whites. The age-adjusted rate for the United States, 1980, was 11.4 (6). Only 8.1% of the excess risk of suicide for Alaskans was due to the high rate of suicide for Alaska Natives (population attributable risk).

Place of Residence

Seventy-three percent of the Native population and 33% of Whites lived in communities of less than 5,000 people. The suicide rate for Natives was almost twice as high in rural areas, while the rate for Whites was slightly higher in urban areas of greater than 5,000 people (Table 3). Similar findings resulted from analysis of the 15- to 24-year-old subgroup of suicides.

Temporal Distribution

For all races combined, the greatest number of suicides occurred in May (N=21) and October (N=20). A roughly bimodal pattern was observed for both White and Native suicides, with peaks in the spring and fall (Figure 3).

The day of injury was known for 185 (95%) of the 195 suicides. Suicides were least likely to occur on Mondays (N=18) with the remainder being nearly equally distributed throughout the week. There was a small peak in suicides among Whites on Tuesdays (N=20) and among Natives on Sundays (N=13). No significant racial or sexual differences were observed in the daily distribution

Table 1. Number and Percent of Suicides by Race, Sex, and 5-Year Age Group Alaska, 1983-1984

	White				Native				All Races*			
	Males		Females		Males		Females		Males		Females	
Age Group	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
<15	1	1.0	0	0.0	1	1.8	0	0.0	2	1.2	0	0.0
15-19	9	8.6	2	8.7	9	16.4	2	20.0	18	11.1	4	12.1
20-24	16	15.2	4	17.4	20	36.4	6	60.0	36	22.2	10	30.3
25-29	16	15.2	4	17.4	12	21.8	1	10.0	29	17.9	5	15.2
30-34	18	17.1	3	13.0	2	3.6	0	0.0	20	12.3	3	9.1
35-39	13	12.4	1	4.3	2	3.6	1	10.0	15	9.3	2	6.1
40-44	9	8.6	4	17.4	0	0.0	0	0.0	10	6.2	4	12.1
45-49	7	6.7	3	13.0	4	7.3	0	0.0	11	6.8	3	9.1
50-54	5	4.8	1	4.3	4	7.3	0	0.0	9	5.6	1	3.0
55-59	2	1.9	0	0.0	1	1.8	0	0.0	3	1.9	0	0.0
60-64	2	1.9	1	4.3	0	0.0	0	0.0	2	1.2	1	3.0
65-69	2	1.9	0	0.0	0	0.0	0	0.0	2	1.2	0	0.0
70-74	2	1.9	0	0.0	0	0.0	0	0.0	2	1.2	0	0.0
75-79	1	1.0	0	0.0	0	0.0	0	0.0	1	0.6	0	0.0
80-84	1	1.0	0	0.0	0	0.0	0	0.0	1	0.6	0	0.0
Unknown	1	1.0	0	0.0	0	0.0	0	0.0	1	0.6	0	0.0
All Ages	105	100.0$^+$	23	100.0$^+$	55	100.0	10	100.0	162	100.0$^+$	33	100.0
Median Age	32.0		33.0		24.0		21.5		28.0		25.0	

*** includes 2 Black males, 26 and 43 years old**

$^+$ columns do not total 100.0% due to rounding artifact

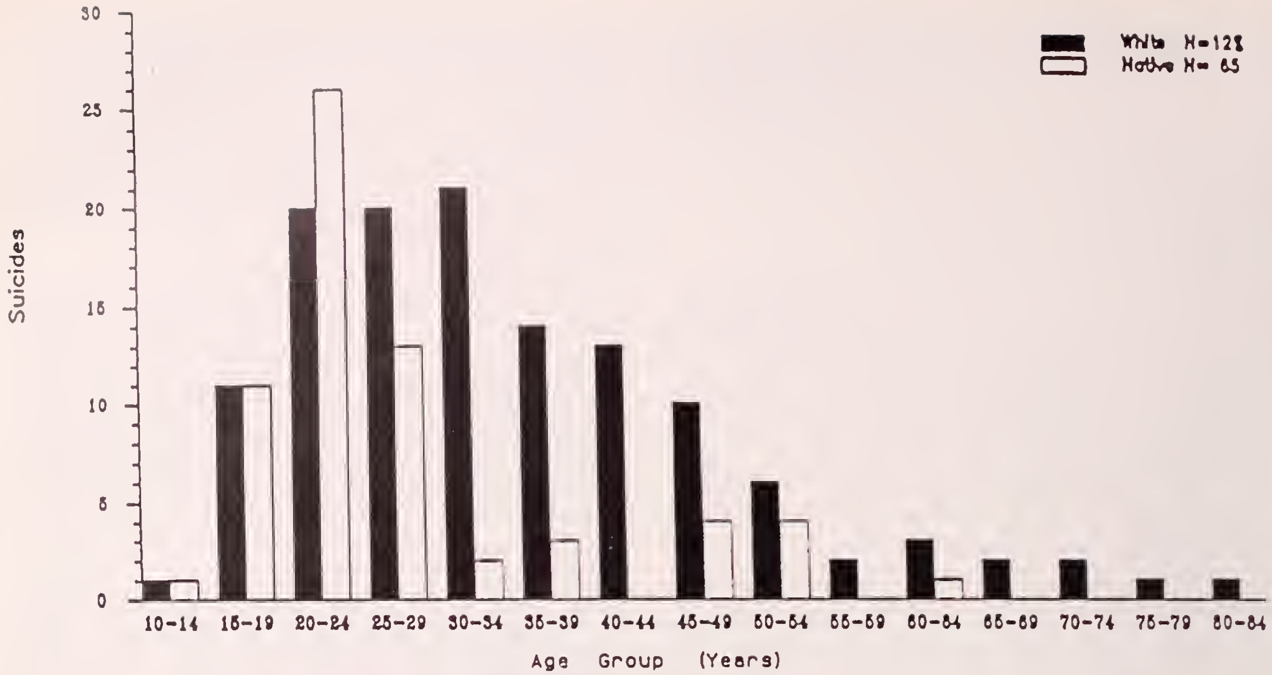

Figure 1: Number of Suicides by Age Group and Race, Alaska, 1983-1984

of suicides.

Suicide Methods

Of 195 suicides, 149 (76%) were the result of gunshot wounds. No significant racial or age differences were observed in the methods used in suicide. The proportion of suicides by overdose was higher among females, and males used a greater variety of methods (Figure 4). Of 187 suicides for which information was available, 120 (64%) occurred at home. There were no significant differences in the frequency distribution of suicide methods between home and elsewhere.

Alcohol

Blood alcohol levels at autopsy were available for 169 (87%) of the 195 suicides. Of those, 99 (59%) had detectable levels of blood alcohol and 53 (31%) had levels greater than 100 mg/dl. Blood alcohol levels by race are shown in Figure 5.

Analysis by multiple logistic regression revealed that suicides with blood alcohol levels greater than or equal to 100 mg/dl were significantly more likely to have died as the result of gunshot wounds ($p < .001$; estimated odds ratio 1.3, 95% confidence interval 1.11-1.47), controlling for the effects of age, race, and sex—none of which were predictive (Table 4).

Table 2. Average Annual Suicide Rates (per 100,000) by Race and Sex Alaska, 1983-1984

	Race		
Sex	White	Native	All Races
Male	25.2	73.5	29.8
Female	6.4	13.9	7.0
Total	16.5	44.2	19.1

Marital Status

Information on marital status was available from death certificates of 189 suicides: 52 (28%) were married; 3 were separated, 36 were divorced, and 2 were widowed, for a total of 41 (22%) previously married; and 96 (51%) were never married. Of 125 suicides in Whites, 44 (35%) were in married Whites, compared to 7 (11%) married among 62 suicides in Natives. This difference was not statistically significant after marital status was adjusted for age ($p > .05$, Mantel-Haenszel chi-square = 3.40).

Employment Status

The "usual occupation...even if retired" was designated "unemployed" on the death certificate of 18 (9.8%) of the 184 suicides for which this information was available. No significant racial differences were observed. A review of testimony in the coroners' records indicated that this is an underestimate of the true unemployment rate for suicides. Average statewide unemployment during the study period was 10.2 percent (7).

Years of Potential Life Lost

The number of years of potential life lost to suicide before age 65 (YPLL) in Alaska during 1983-1984 was 6,407. Because suicides in Natives occurred at younger ages, they accounted for a disproportionately larger

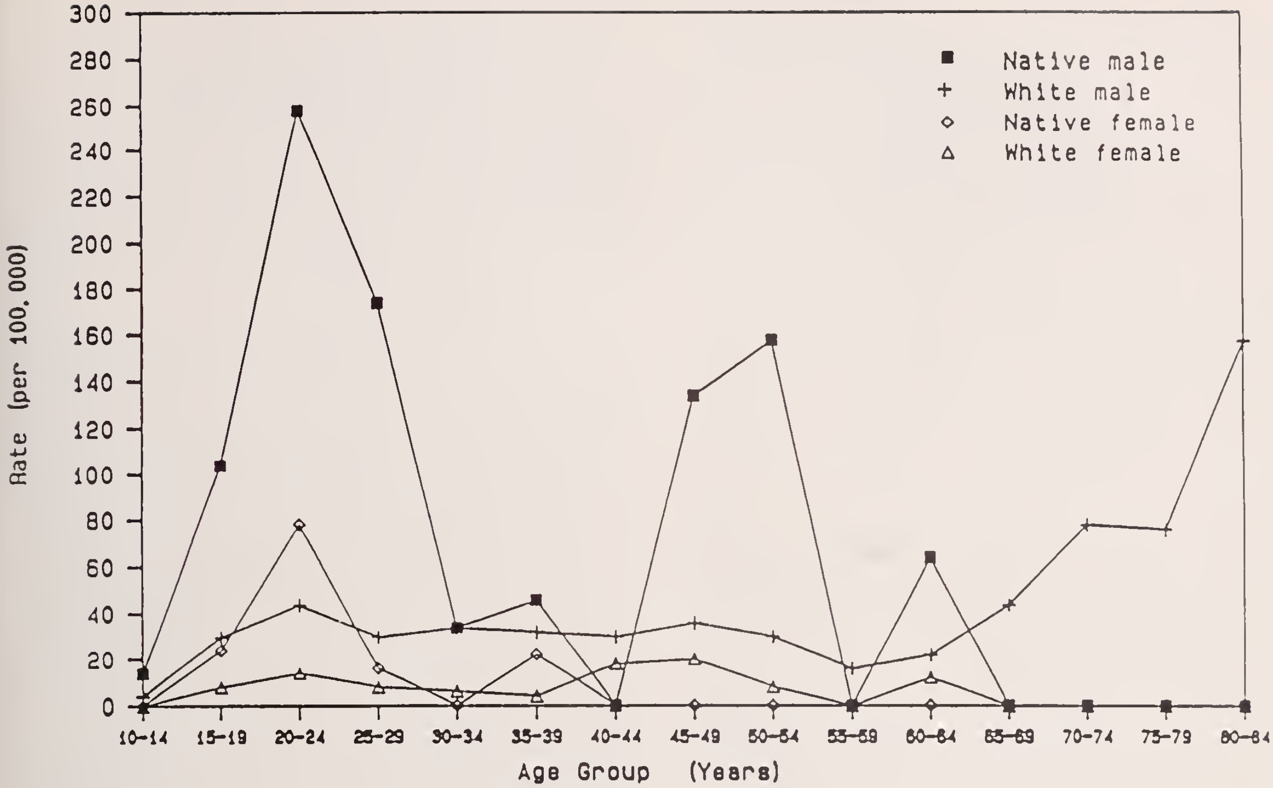

Figure 2: Rate of Suicide by Age, Race, and Sex, Alaska, 1983-1984

share of the total YPLL. Natives accounted for 65 (35%) of the 188 suicides before age 65 and 2,476 (39%) of the YPLL.

The average annual rate of YPLL statewide was 629 per 100,000 population. For Natives, the rate was 1,684 YPLL per 100,000 population per year, 3.4 times the annual rate for Whites—499.0 YPLL per 100,000. The rate of YPLL to suicide in the United States in 1983 was 270.1 (8).

DISCUSSION

As with most descriptive studies, this study of suicide in Alaska raises more questions than it answers. Because of differences in methodology, our results are not directly comparable to those obtained from studies based solely upon state and national vital records. In another report (4), we demonstrated that our data were more accurate and that errors in official records during 1983-1984 resulted almost exclusively from underrecording of suicides. These errors were not randomly distributed. Suicides from rural, predominately Native areas more often failed to appear in the state and national records.

Alaskans committed suicide more frequently and at younger ages than were observed in recent national statistics. The age-adjusted rate of suicide in Alaska, 1983-1984, was almost twice the rate observed for the total United States in 1980. For Native Alaskans, it was four times the national rate. The median age among all suicides in Alaska was 12 years younger than the median age of suicides in the United States in 1980 (6). For Native Alaskans, the median age of suicides was 17 years younger than that observed nationally.

The observation of a high rate of suicide for young Alaskans appears to be an exaggeration of the changing pattern of suicides noted in national statistics from the United States and elsewhere (1,9-12). Suicide no longer is an affliction primarily of the elderly, but a growing epidemic among the young. The findings on the number and rate of years of potential life lost to suicide are a statistical reflection of the tragic impact suicide is having among young Alaskans.

The trend toward rising rates of suicide in the younger age groups has been most evident in populations experiencing rapid change and social disorganization, a condition which has been used to characterize the Native Alaskan culture, other North American Indians, and much of the developing world (12-16). This study did not address complex social issues, but the need for improved understanding is clear.

Seiden (1) observed a strong inverse relationship between population density and the suicide rate for young people in the western United States. We observed a similar relationship for Natives, but not for Whites in Alaska.

Depression, unemployment, and alcoholism have also been cited as important contributing factors to suicide (17-21). This study did not examine emotional status, and our findings relating to employment may not be reliable because they were based only upon death certificate data. Employment in Alaska is often seasonal—

Table 3. Crude Suicide Rates per 100,000 per Year by Race and Residence Alaska, 1983-1984

Race	Residence Urban*	 Rural	 Total
Native	27.4	50.5	44.2
White	15.2	14.4	14.9
All Races	15.9	24.3	19.1

* > 5,000 population

leaving the nominally employed idle for months at a time. But we did observe a high prevalence of acute alcohol intoxication among Alaskan suicides, especially Natives, based upon highly reliable autopsy data. Whether alcohol was a causative factor, or whether acute intoxication reflected chronic alcoholism was not answered by this study.

The findings regarding marital status serve as a useful baseline, but the absence of comparison data from the general population or appropriate control groups does not allow conclusions about possible causality.

This study did not address many other social and economic issues which may have contributed to the high rate of suicide in Natives. During the past fifty years, Alaska Natives have gone from a majority population to a minority, from a subsistence economy to a cash economy, and through extreme cycles of economic boom and bust. These and many other social, cultural, and economic changes have resulted in profound changes in family roles and community function, and traditional cultural values and support systems have been severely undermined (14,15).

Recent descriptions of seasonal affective disorder (SAD) suggest that the long, dark Alaskan winters may contribute to the high rate of suicide (22,23). The incidence peaks which we observed in the spring and fall are remarkably consistent with observations from many different latitudes within the northern hemisphere, but they do not rule out SAD as a contributing factor in at least some suicides (24).

The low incidence of suicides on Mondays among Alaskan suicides during 1983 and 1984 contrasts with the Monday peak incidence described for all U.S. suicides, 1972-1976 (25). This was based upon date of injury data, not date of death. The reasons for this discrepancy are not clear, but may be related to employment or other social factors, such as drinking behavior.

The availability of firearms has been hotly debated as a contributing factor to the increasing incidence of suicide among young people (11,26-29). Indeed, firearms are widely present and easily available in Alaska, and the large majority of suicides in our study were the result of gunshot wounds, especially when the victim was acutely intoxicated with alcohol. These observations are not sufficient to prove causality, but they do invite further research.

Others have observed a trend among female suicide victims to more frequently choose death by firearms over the "traditional" method of drug overdose (6,29, 30). Our study shows that in Alaska they have reached parity with their male counterparts in this regard, yet females still represented only a small proportion of completed suicides. This study did not examine nonfatal suicide attempts.

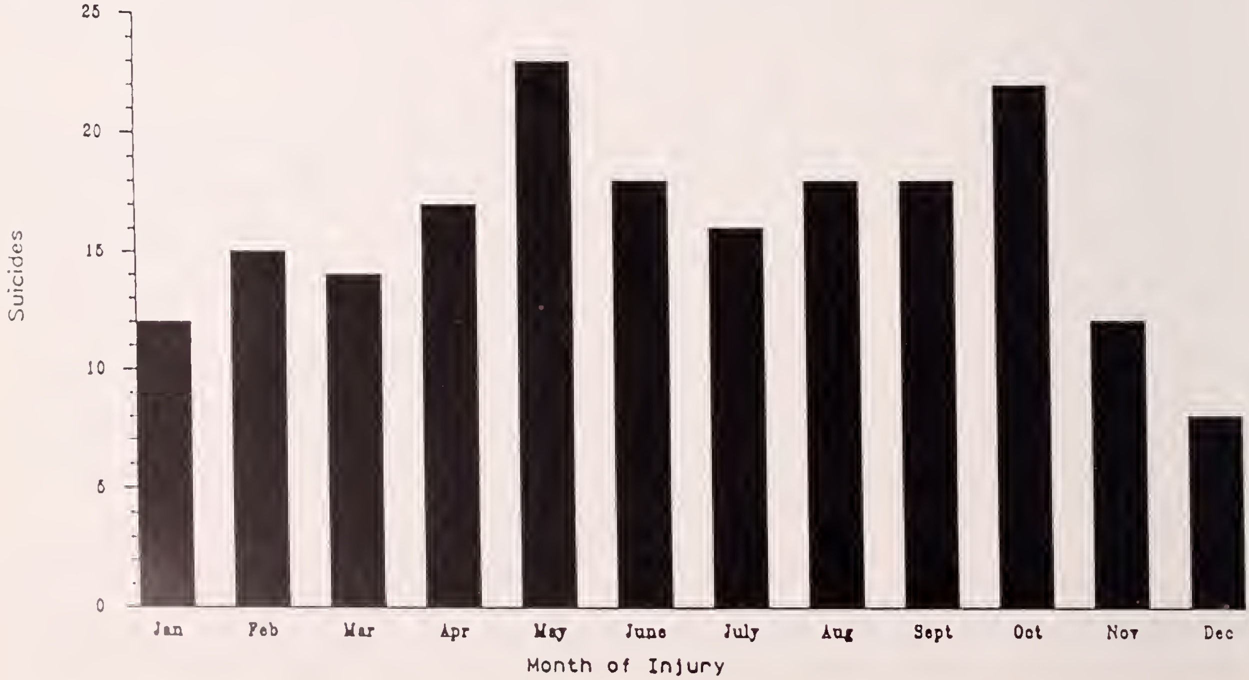

Figure 3: Month of Injury for Suicides, Alaska, 1983-1984

Table 4. Regression of Firearm Suicide by Race, Sex, Age and Blood Alcohol Level Alaska, 1983–1984• (N=168)

Regressor	Regression Coefficient
Intercept	0.727 *
Race	-0.043
Sex	-0.120
Age	0.004
Blood Alcohol $\geq$ 100 mg/dl	0.245 *+
$R^2 = 0.096$	

* Coefficient is at least twice its standard error ($p < .05$).

+ $p = .0008$; estimate of odds ratio = 1.3, 95% confidence interval 1.11–1.47.

• Age was a continuous variable. Race, sex, and blood alcohol were dummy variables:
Native = 1, non–Natives = 0
Male = 1, female = 0
Blood alcohol $\geq$ 100 mg/dl = 1
Blood alcohol $<$ 100 mg/dl = 0

The fact that suicides occurred more frequently and at younger ages among Alaska Natives than among Whites identifies a high–risk population in need of intervention. Most suicides in Alaska are impulsive (3,31), occur at home, and involve alcohol and firearms. Opportunities for immediate crisis intervention, therefore, seem limited and doomed to failure. We believe a more effective strategy for prevention is to target the highest risk cohorts—rural Alaska Native males aged 10–24—for preventive education and social support. Education should encourage expression of emotions, and recognition of depression and the need for help. Men's support groups could reduce the great pressures on young males and provide help when needed.

Additional research on non–fatal suicide attempts (parasuicide), and on the differences between communities with very high and very low suicide rates would be helpful in formulating more specific plans for an effective public health response to the problem of suicide in Alaska.

ACKNOWLEDGEMENTS

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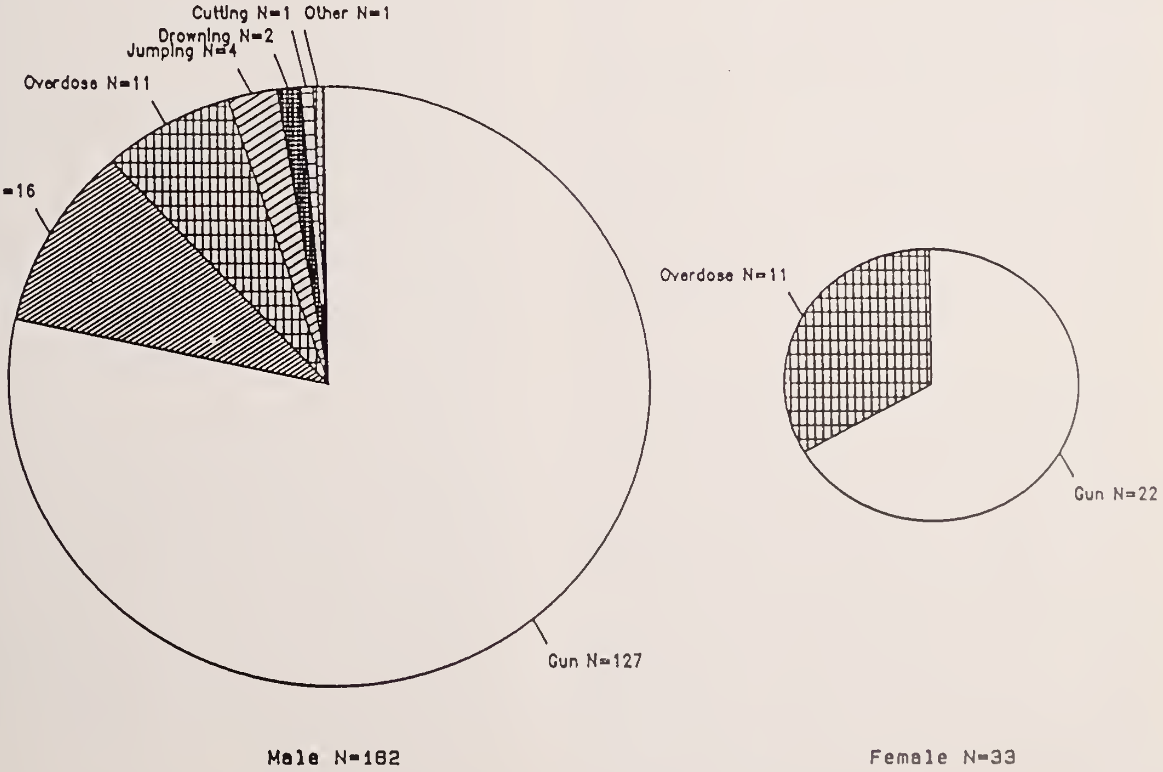

Male N=182

Female N=33

Figure 4: Methods of Suicide by Sex, Alaska, 1983–1984

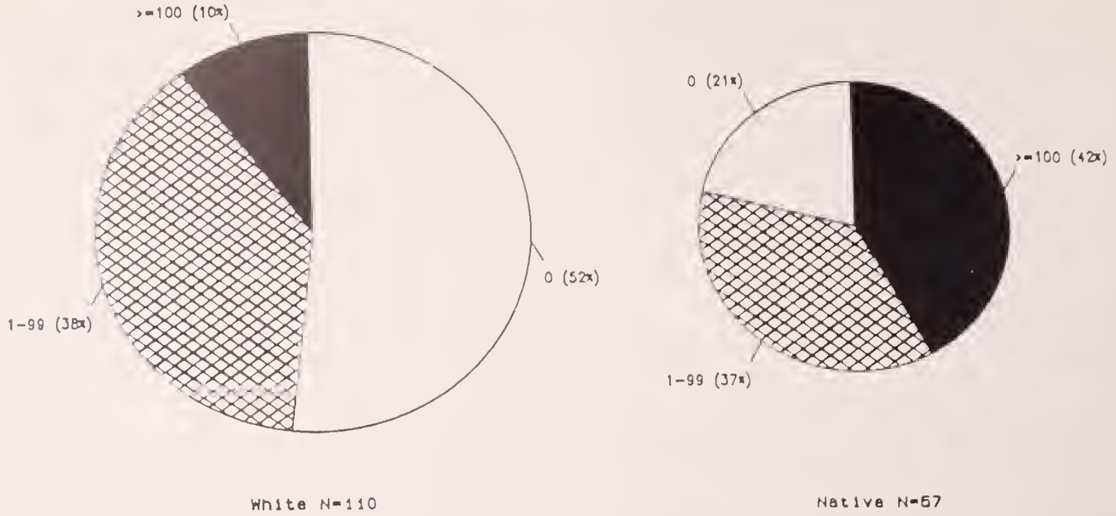

Figure 5: Blood Alcohol Levels at Autopsy of Suicides by Race, Alaska, 1983–1984

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AIDS and the Physician: A Policy Crisis

by Ann N. James, Ph.D., J.D.[1]

ABSTRACT

Because Acquired Immunodeficiency Syndrome affects everyone, the disease has become a major challenge to the health care industry. Specific policies and protocols must be drafted to ensure that important issues are addressed in a consistent and careful manner, thereby enhancing the safety of all concerned and protecting the rights of both patients and health care providers. Physicians must work within their own profession as well as with hospital administrators in considering such issues as universal precautions, testing procedures, and patient confidentiality. Medical staff members must evaluate appropriate methods of dealing not only with AIDS patients but with fellow practitioners and other health care professionals who contract the disease. Hospital and medical staff policies should be drafted to meet the individual needs of each health care facility.

Acquired Immunodeficiency Syndrome ("AIDS") has become the great challenge to the health care system and all providers. "AIDS is, without a doubt, one of the most devastating infectious diseases the world has ever known"(1). Both in private practice and as a member of a hospital medical staff, the physician faces another kind of challenge: the development of policies to address AIDS in the health care setting. It is apparent from recent publicity that if physicians do not meet this challenge, a government regulator is likely to impose guidance externally. The purposes of this article are to discuss a conceptual approach to development of an AIDS policy for a health care facility and to address issues such as universal precautions, patients' rights, and the medical staff member with AIDS. Alaska, like other states, has a framework of statutes and regulations to which an AIDS policy must conform, but the focus of this article is on the *concepts* to be addressed in an AIDS policy rather than on legislative parameters.

AIDS policies do not lend themselves to prepackaged, standard forms. Each medical staff, together with its hospital's board, administration, and other professionals must develop an appropriate policy for that hospital's unique needs. To be effective, the policy must evolve within the institution.

Universal blood and body fluid protocols have been recommended for *all* patients by the Centers for Disease Control (CDC) in guidelines published on August 21, 1987 (2). Taking a patient's medical history and performing a physical examination cannot reliably identify all patients infected with human immunodeficiency virus (HIV) or other blood-borne pathogens, such as the hepatitis B virus. Consequently, strict blood and body fluid protocols must be followed in all patient encounters. Adhering to universal protocols is especially critical for those physicians and health care workers in emergency care settings where the risk of exposure to blood is increased and the infectious status of the patient is typically not known.

Recently the Occupational Safety and Health Administration (OSHA) announced that it will inspect hospitals to ensure that health care workers are protected against blood-borne diseases such as AIDS and hepatitis B. Although OSHA is initially reviewing hospitals under its general regulations for employees who work with toxic substances, specific regulations for the protection of health care workers are being developed and are expected to become final within two years. Draft OSHA regulations are based on the CDC guidelines. Upon receipt of a complaint filed by an employee alleging unsafe blood or body fluid handling procedures, OSHA will investigate the hospital. If protective equipment is not being furnished to employees at risk or if the equipment is not utilized in the proper manner according to OSHA regulations, OSHA has the authority to assess a fine against any employer found to be violating such regulations (3). In addition to his responsibility as a member of the medical staff to pursue compliance with the OSHA regulations in the hospital setting, the physician must consider his duty to assure that his office employees are afforded the protections offered by compliance with OSHA regulations (4).

Patient care issues are central to the development of any AIDS policy. Although the principal concern is the actual care rendered to an AIDS patient, testing of all patients and confidentiality of the results and diagnosis are also critical issues. The long incubation period between exposure to HIV and development of measurable antibodies means that a negative HIV test result does not rule out the presence of infection. Thus, concern has been expressed by almost every national health care organization, particularly the American Hospital Association, that reliance on HIV testing as an indicator of exposure to, and infection with, HIV could create an unnecessary risk for health care professionals. Other problems are inherent in the enzyme-linked immunosorbent assay (ELISA) test. False positive and false negative test results are possible and often only the more expensive "western blot" test can accurately determine whether a person has been infected with HIV (5). Today, hospitals generally have an AIDS testing policy that is unwritten and unstated. The hospitals simply rely on the medical judgment of physician members of its medical staff to order tests as appropriate. Unfortunately, such an approach does not serve either the physician, the facility, or the patients well.

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Some hospitals, like Vanderbilt University Hospital in Nashville, Tennessee, test only surgical patients for the HIV antibody. Others, such as Vanderbilt's neighbor, St. Thomas Hospital, have instituted and then retracted universal testing programs (6). At least one hospital, The Methodist Hospital in Houston, Texas, has established a "voluntary" program. At The Methodist Hospital, each patient presented for admission will be offered the opportunity to be tested for the HIV antibody. Those declining to be tested will be treated as though they are diagnosed as HIV positive (i.e., universal blood and body fluid precautions will be observed) but will not otherwise be subject to discrimination. Each patient bears the responsibility to pay for the test (7). While The Methodist Hospital policy has been publicized, final details are not yet fully developed and, as with any innovative policy, adjustments in the implementation of the policy will be required. As a result of the publication of The Methodist Hospital's proposed policy, some patient advocates are inquiring about the HIV antibody status of the physicians and staff.

Some organizations have suggested a mandatory testing policy if voluntary testing programs fail to curb the rate of HIV infection (8). The problems inherent in a mandatory program are significant, especially for emergency care and for patient access to care. For example, a mandatory testing policy would have to address whether patients presenting in the emergency department would be routinely tested, and if so, how consent would be obtained, how counseling would be conducted, and how test results would be reported in a timely and effective manner. Moreover, a mandatory testing policy would have to address access issues, such as a patient's inability to pay for the test and a patient's reluctance to seek health care for fear of being tested.

The key policy issues to address for a testing program involve who should be tested, where testing should be done, and how pre- and post-testing counseling will be provided. In addition, the members of the medical staff should jointly decide how and when, as a general matter, a patient should be informed about positive HIV test results and how and when spouses, significant others, or family members might be informed.

Patient confidentiality is another critical issue. The state may establish specific confidentiality requirements for AIDS patients, but *every* patient is entitled to an expectation of privacy with regard to his medical care and treatment (9). The need for the health care worker to know of potential deadly risks must be balanced against the patient's right for privacy. The American Medical Records Association (AMRA) has recommended that HIV antibody test results be recorded only in the patient's chart and not placed in a separate report or file (10). AMRA recommends that the chart of a hospitalized patient who has tested positive for HIV exposure should not generally be available to hospital personnel but rather should be stored in a less accessible location such as the nursing station. Development of a policy that addresses confidentiality can create an understanding among all staff of the need to maintain the privacy of each patient.

The health care community's concerns have focused on the issue of testing patients for exposure to HIV to the extent that another, equally important consideration has often not been confronted. This somewhat neglected consideration concerns the type of consent that should be obtained from patients who are likely to receive a blood transfusion. Even though the nation's blood supply has been carefully monitored since the development of the ELISA test, the extremely protracted period of time from exposure and infection to the appearance of HIV antibody indicates that transfusion-related infections are still possible. At least one such case of transfusion-associated infection was reported after universal blood testing was initiated (11).

Autologous donations should generally be advised for elective surgeries. Should directed donations be offered as another alternative? The American Association of Blood Banks does not recommend directed donations (12). One federal district court in the case of *Kozlup vs. Georgetown University* (13), found that informing a patient of the alternative of directed blood donations was not part of a community standard for health care, and hence, failure to inform was not malpractice. However, the present information on the risks of infection with HIV associated with blood transfusions, from 1 in 48,000 units to 1 in 1,000,000 units, would suggest that patients should be informed of the alternative of directed donation (14). Illinois, for example, has amended its blood banking act to allow designated donations (15).

Organ transplant action is another source of possible exposure to HIV and a risk of which patients should be informed even though the alternative, should the transplant be refused, may be death. At least one case of HIV infection by means of an organ transplant occurred as a result of a donor who, originally tested as seronegative, converted during the time of his terminal illness possibly due to transfusions that he received. The three organs transplanted resulted in seroconversion by two of the recipients and the third organ transplant recipient died of another illness (16). When transplant candidates are awaiting receipt of donor organs, particularly when the medication condition is chronic, they or their families may be unable to address the risk of HIV infection nationally. On the other hand, if information relating to the risks of HIV exposure is presented by the physician to the patient at the time of entry into a transplant program, each patient or the responsible family member should be better able to make an informed decision.

Finally, the most sensitive issue in the development of an AIDS policy is to address one's own profession and the profession's response to the disease. Two particular concerns face physicians: the seropositive practitioner and the practitioner who will not treat AIDS patients (17). At the least, some consensus of the medical staff should be reached on each of these issues.

If a physician becomes seropositive, whether that fact should be disclosed on the medical staff application for appointment or reappointment becomes a dilemma for the practitioner, since such disclosure may lead to disaster in his personal life and to loss of his practice (18). The seropositive practitioner who wants to continue to work with his colleagues and can assure that he can provide the best patient care while he is only seropositive should be allowed to continue on the medical staff. Further, a practitioner who has AIDS or AIDS-related complex (ARC) should not be restricted from continuing to serve on the medical staff unless otherwise medically contraindicated. If the seropositive practitioner has AIDS and has had some illness but wants to return to his practice, then the CDC guidelines suggest that the physician, his physician, the medical director of the hospital, and the administrator should meet and mutually determine that seropositive physician is able to return to his practice (18). Without some clear policy, this circumstance can lead to acrimony, adverse publicity, and poor policy made under adverse circumstances.

To whom can the seropositive practitioner disclose his condition without waiving his own right to privacy and confidentiality? A medical staff must review its own policies, guidelines, bylaws, and committee responsibilities to determine how such information can be handled confidentially. The alternative is that the seropositive practitioner will have little choice but to carry the secret alone. The experiences of many, including the Dallas pediatrician who was driven from his practice because of hysteria surrounding the publicity of his seropositive condition, demonstrate that a viable and confidential alternative must be developed (19).

The practitioner who is reluctant to treat AIDS patients says, "'I've got to be selfish...I've got to think about myself; I've got to think about my family. That responsibility is greater than to the patient'" (20). Other practitioners note the poor prognosis of AIDS patients and the responsibility that the surgeon carries for the safety of the operating room team as reasons not to provide treatment for AIDS patients (21). Given the delay in development of detectable antibodies in many patients, all practitioners and health care workers must render care for each patient as if that patient were seropositive for HIV. Thus, the reluctant practitioner may believe that his behavior protects his own family, but the risk of accidental exposure remains as long as he continues to deliver health care.

By refusing to treat any known AIDS patients, a practitioner increases the risk of accidental exposure for other physicians practicing at the same hospital or in the same specialty. Such behavior perpetuates the stigma of AIDS and compromises the physician's own ethical commitment. The refusal to treat AIDS patients is a sensitive question to address within the medical staff, but it is unfair to the practitioners who continue to provide treatment to all patients not to determine, as a medical staff, what the AIDS treatment policy should be. If existing bylaws, rules, and policies are not sufficient to address the situation of a practitioner who refuses to treat HIV-positive or AIDS patients, then an appropriate policy or revision of the bylaws should be considered.

Risk, testing, confidentiality, consent, precautions—AIDS can never be treated as someone else's problem in health care, because AIDS affects everyone. The medical community is challenged on every front by AIDS, but development of a comprehensive AIDS policy also offers an opportunity to establish a policy that is sensitive to the needs of the patient and the practitioners, that enhances the understanding of these issues for physicians and staff, and that addresses the most critical public health concerns confronting medicine today.

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The Alaska Public Health Association invites you to participate in the 1988 Health Congress by presenting a paper. ALPHA encourages people involved in public health in Alaska to answer this call for papers.

The 1988 Health Congress will be held April 25–27 at the Sheraton Anchorage, and the theme is "PUTTING THE PUBLIC IN PUBLIC HEALTH." Therefore, we urge all potential presenters to highlight the role of public involvement in the process and/or the impact of findings upon the public when submitting abstracts for consideration.

Papers will be presented in concurrent sessions designed to represent a wide variety of Alaska public health disciplines. Topics may include maternal/child health, epidemiology, nutrition, sanitation, AIDS, health education, health administration, environmental health, health planning, mental health, occupational health and safety, injury control, alcohol problems/prevention, and self-help groups.

ALPHA looks forward to providing a forum for sharing current knowledge and work experiences. We especially want to hear from community-based and rural providers as well as urban and state members.

Completed abstracts of papers and presentation applications must be submitted by January 30, 1988. Please include five copies of your abstract. The abstract should be a brief summary (no more than 200 words) of your presentation. Send applications and copies of your abstracts to:

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If you have questions, please call Jan Wills in Anchorage at 343-4800, or DeLen Doutrick in Dillingham at 842-2276. We look forward to hearing from you.

CARAFATE®
(sucralfate)

BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate.

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that the simultaneous administration of CARAFATE with tetracycline, phenytoin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. The clinical significance of these animal studies is yet to be defined.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No evidence of drug-related tumorigenicity was found in chronic oral toxicity studies of 24 months' duration conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies have not been conducted.

Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients, adverse effects were reported in 121 (4.7%). Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED
CARAFATE (sucralfate) 1-gm pink tablets are supplied in bottles of 100 and in Unit Dose Identification Paks of 100. The tablets are embossed with MARION/1712.
Issued 3/84

References:
1. Grossman MI: *Scand J Gastroenterol* 58 (suppl 15):7-16, 1980.
2. Marks IN, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 70-81, 1984.
3. Krentz K, Jablonowski H, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 62-69, 1984.

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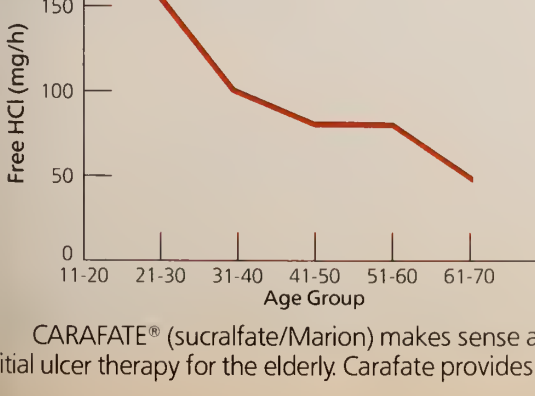
Specialized ulcer therapy

When advancing age signals reduced acid secretion

If your duodenal ulcer patient is over 55, decreased mucosal resistance is more likely to cause an ulcer than hypersecretion of acid-pepsin.[1] A tendency toward lower acid secretion with advancing age has been shown.[2,3]

Declining gastric secretion and age[3]



CARAFATE® (sucralfate/Marion) makes sense as initial ulcer therapy for the elderly. Carafate provides ulcer healing rates comparable to H_2 antagonists without the risk of systemic side effects or drug interactions—an important benefit for older patients.

The unique, nonsystemic action of Carafate enhances the body's own ulcer healing ability, strengthening the mucosal structure as it protects damaged tissue from further injury.

When advancing age signals reduced acid secretion, choose the specialized ulcer therapy of safe, nonsystemic Carafate.

Nothing works like

CARAFATE®
sucralfate/Marion

Please see adjoining page for references and brief summary of prescribing information.

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There's never been a better time for he

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Proven benefits beyond relief of vasomotor symptoms

No other estrogen proven effective for osteoporosis

Only conjugated estrogens tablets have established efficacy in both osteoporosis[1] and vasomotor symptoms* at 0.625 mg/day. No other estrogen, oral or transdermal, has established clinical evidence or minimum effective dose in both indications.

No estrogen proven safer

PREMARIN is the most extensively tested estrogen, with an unsurpassed record of long-term safety.
And clinical evidence shows a significantly reduced risk of endometrial hyperplasia when cycled with a progestin.[2]

PREMARIN®
(conjugated estrogens tablets)

Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms

Please see following page for brief summary
of prescribing information.

For moderate-to-severe vasomotor symptoms and for osteoporosis

PREMARIN®
(conjugated estrogens tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

The appearance of these tablets is a trademark of Ayerst Laboratories.

For atrophic vaginitis

PREMARIN®
(conjugated estrogens)

Vaginal Cream

0.625 mg/g

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

PREMARIN® Brand of conjugated estrogens tablets, USP
PREMARIN® Brand of conjugated estrogens Vaginal Cream, in a nonliquefying base

1 ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA
Three independent, case-controlled studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year This risk was independent of the other known risk factors for endometrial cancer These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade The three case-controlled studies reported that the risk of endometrial cancer in estrogen users was about 4 5 to 13 9 times greater than in nonusers The risk appears to depend on both duration of treatment and on estrogen dose In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible When prolonged treatment is medically indicated, the patient should be reassessed on at least a semi-annual basis to determine the need for continued therapy Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration, it therefore appears prudent to utilize such a regimen Close clinical surveillance of all women taking estrogens is important In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding adequate diagnostic measures should be undertaken to rule out malignancy There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equi-estrogenic doses
2 ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring It has been shown that females exposed in utero to diethylstilbestrol, a nonsteroidal estrogen, have an increased risk of developing, in later life, a form of vaginal or cervical cancer that is ordinarily extremely rare This risk has been estimated as not greater than 4 per 1,000 exposures Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix Although these changes are histologically benign, it is not known whether they are precursors of malignancy Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb-reduction defects One case-controlled study estimated a 4 7-fold increased risk of limb-reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment The data suggest that the risk of limb-reduction defects in exposed fetuses is somewhat less than 1 per 1,000 In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well-controlled studies that progestogens are effective for these uses If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation

DESCRIPTION: PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0 3 mg, 0 625 mg 0 9 mg, 1 25 mg, and 2 5 mg strengths of conjugated estrogens Cream is available as 0 625 mg conjugated estrogens per gram

INDICATIONS AND USAGE: PREMARIN (conjugated estrogens tablets, USP) Moderate-to-severe *vasomotor* symptoms associated with the menopause (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions) Osteoporosis (abnormally low bone mass) Atrophic vaginitis Kraurosis vulvae Female castration

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae

PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING)

Concomitant Progestin Use: The lowest effective dose appropriate for the specific indication should be utilized Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes Whether this will provide protection from endometrial carcinoma has not been clearly established There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens (See PRECAUTIONS) The choice of progestin and dosage may be important, product labeling should be reviewed to minimize possible adverse effects

CONTRAINDICATIONS: Estrogens should not be used in women (or men) with any of the following conditions. 1 Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease 2 Known or suspected estrogen-dependent neoplasia 3 Known or suspected pregnancy (see Boxed Warning) 4 Undiagnosed abnormal genital bleeding 5 Active thrombophlebitis or thromboembolic disorders 6 A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy)

WARNINGS: Estrogens have been reported to increase the risk of endometrial carcinoma (see Boxed Warning) However a recent large case-controlled study indicated no increase in risk of breast cancer in postmenopausal women A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use They should be used with caution in patients with cerebral vascular or coronary artery disease Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism, and thrombophlebitis When doses of this size are used any of the thromboembolic and thrombotic adverse effects should be considered a clear risk

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness abdominal mass, or hypovolemic shock Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives For this reason, diabetic patients should be carefully observed Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases

PRECAUTIONS: Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs and should include a Papanicolaou smear As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed Conditions influenced by fluid retention, such as asthma, epilepsy migraine, and cardiac or renal dysfunction, require careful observation Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding mastodynia, etc Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients Oral contraceptives appear to be associated with an increased incidence of mental depression Patients with a history of depression should be carefully observed Pre-existing uterine leiomyomata may increase in size during estrogen use The pathologist should be advised of estrogen therapy when relevant specimens are submitted If jaundice develops in any patient receiving estrogen the medication should be discontinued while the cause is investigated Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia or in young patients in whom bone growth is not yet complete If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism

The following changes may be expected with larger doses of estrogen

a Increased sulfobromophthalein retention

b Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3, increased norepinephrine-induced platelet aggregability

c Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T_4 by column, or T_4 by radioimmunoassay Free T_3 resin uptake is decreased reflecting the elevated TBG free T_4 concentration is unaltered

d Impaired glucose tolerance

e Decreased pregnanediol excretion

f Reduced response to metyrapone test

g Reduced serum folate concentration

h Increased serum triglyceride and phospholipid concentration

As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk

Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver However, in a recent, large case-controlled study of postmenopausal women there was no increase in risk of breast cancer with use of conjugated estrogens

ADVERSE REACTIONS: The following have been reported with estrogenic therapy including oral contraceptives breakthrough bleeding spotting change in menstrual flow dysmenorrhea, premenstrual-like syndrome, amenorrhea during and after treatment, increase in size of uterine fibromyomata, vaginal candidiasis, change in cervical erosion and in degree of cervical secretion, cystitis-like syndrome, tenderness, enlargement, secretion (of breasts), nausea, vomiting, abdominal cramps, bloating, cholestatic jaundice, chloasma or melasma which may persist when drug is discontinued, erythema multiforme, erythema nodosum, hemorrhagic eruption, loss of scalp hair, hirsutism, steepening of corneal curvature, intolerance to contact lenses, headache migraine, dizziness, mental depression, chorea, increase or decrease in weight, reduced carbohydrate tolerance aggravation of porphyria, edema, changes in libido

ACUTE OVERDOSAGE: May cause nausea, and withdrawal bleeding may occur in females

DOSAGE AND ADMINISTRATION:

PREMARIN® Brand of conjugated estrogens tablets, USP

1 *Given cyclically for short-term use only* For treatment of moderate-to-severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0 3 mg to 1 25 mg or more daily) The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible Administration should be cyclic (eg, three weeks on and one week off) Attempts to discontinue or taper medication should be made at three- to six-month intervals

2 *Given cyclically* Osteoporosis, Female castration Osteoporosis —0 625 mg daily Administration should be cyclic (eg, three weeks on and one week off) Female castration—1 25 mg daily, cyclically Adjust upward or downward according to response of the patient For maintenance, adjust dosage to lowest level that will provide effective control

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding

PREMARIN® Brand of conjugated estrogens Vaginal Cream

Given cyclically for short-term use only For treatment of atrophic vaginitis or kraurosis vulvae

The lowest dose that will control symptoms should be chosen and medication should be discontinued a promptly as possible

Administration should be cyclic (eg, three weeks on and one week off)

Attempts to discontinue or taper medication should be made at three- to six-month intervals

Usual dosage range: 2 g to 4 g daily, intravaginally, depending on the severity of the condition

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer an appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurrin abnormal vaginal bleeding

References:

1. Lindsay R, Hart OM, Clark OM The minimum effective dose of estrogen for prevention of postmenopaus bone loss *Obstet Gynecol* 1984,63 759-763 **2.** Studd JWW, Thom MH, Paterson MEL, et al: The prevention an treatment of endometrial pathology in postmenopausal women receiving exogenous estrogens, in Pasetto N Paoletti R Ambrus JL (eds) *The Menopause and Postmenopause* Lancaster, England, MTP Press Ltd, 198 chap 13

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Special Feature

An Alternate Proposal for Compensating Injuries Occurring in the Health Care Delivery System

by Carl L. Wedekind, Jr., LL.B.[1]

ABSTRACT

This paper proposes adopting a no-fault approach to compensating injuries occurring in the delivery of health care, similar to the original approach under the Workers' Compensation system applying to injuries occurring in the work place. The proposal is based on the statistical evidence that over 60% of all the dollars spent on medical malpractice cases are spent to pay costs and attorney fees to determine fault.

The proposed Patients' Compensation system would determine claims administratively by a 15-member Board, assisted by an Advisory Board of medical specialists, which would also serve as a peer review board of all incidents where medical negligence is indicated.

The plan would compensate injuries on the basis of lost wages and medical expenses, but would exclude injuries which were inherent risks of the treatment modality and would still require proof of negligence where the injury results in death.

A statistical review of the costs of 100 closed claims under the current tort system compared with similar costs under the proposed Patients' Compensation system discloses that savings of almost 50% would be achieved under the Patients' Compensation system with the injured party receiving more net benefits than he is now receiving under the current tort system.

Tort reform is being seriously debated and acted upon in many of the State Legislatures and in the Federal Congress. Proposals vary but frequently break down to the categories of a) limiting the amount of recovery, b) regulating contingency fees for attorneys, c) allowing credit for collateral source payments available to the injured party, d) providing for periodic payments rather than lump sum award, e) establishing economic incentives to promote a prompt resolution, and so forth (1). The nagging question keeps surfacing, "Will any of these measures help in the availability and cost of liability insurance?", and "Are these proposals fair?"

This proposal addresses a system of reform which will, if carried out properly, help directly in both the cost and availability of professional liability insurance for physicians, hospitals, and other health care providers and is intended to contain the basic tenets of fairness. The proposal is the result of several years of research and review and embodies a number of ideas from different sources and the thoughts and experiences of the author (2). No claim of originality is made unless it be in the perceived uniqueness of the overall plan.

This proposal relates only to medical professional liability insurance problems, because of the conviction that the area of medical malpractice is unique in the tort system and should be dealt with separately from other liability insurance problems.

The basic assumptions that form the framework of the proposal are these:

1. Persons who are injured in the health care system should be compensated.
2. Persons, or institutions, responsible for such injuries should be held accountable.
3. Provable fault should not be a requisite to recover compensation for an injury other than death.
4. The inherent identifiable risk of any modality of treatment should be excluded from coverage, and that risk insured through a separate mechanism.
5. The current tort system for determining medical malpractice has a serious economic flaw as the majority of all claims dollars are being spent to determine the question of fault.

The statistics that led to this proposal are the results of closed claims studies done by the Kentucky Medical Insurance Company (KMIC) a physician owned medical malpractice company. These statistical studies are similar in results to closed claims studies done by other medical malpractice carriers (3).

The 30-month period covered by this study is from January 1, 1984 to June 30, 1986, and includes all medical malpractice claims closed by KMIC in that time span.

Of the 268 closed claims studied it was determined that in 112 cases (42%) there was liability. That is, there was a recognized standard of care that had not been met.

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[1] Reprinted with permission from A. M. Best Co., Best's Review, May 1987. Published also in the Journal of the Kentucky Medical Association, 1987;June:317-322.

The average indemnity payment to the plaintiff for all 112 liability cases was $53,367. The average cost to defend these 112 cases (defense lawyers, expert witness, depositions, court costs, etc.) was $8,166 for a total average cost on liability cases of $61,533.

On those cases where we, or a judge or jury, determined there was no liability (156 cases, 58%), there were zero indemnity payments made and the average defense cost per claim was $5,304.

The total 268 claims cost, for both indemnity payments and defense costs, was $7,845,679. The breakdown is as follows:

Indemnity payments	$5,977,065
Defense costs	1,868,614
Total	$7,845,679

The indemnity payments are made directly to the plaintiff and his or her attorney. We are advised that the average contingent attorney's fee on medical malpractice cases in the Kentucky area currently is 40%, and the plaintiff's expert witness, depositions, and other costs are on top of that and can be currently estimated at about 10%. So the plaintiff receives approximately 50% of the indemnity settlement (4).

Thus the use of the $7,845,679 total paid out on these 268 claims breaks down as follows:

To the Plaintiff	$2,988,532	38%
To the Plaintiff's attorney and costs	2,988,532	38%
To the Defendant's attorney and costs	1,868,614	24%
Total	$7,845,614	100.0%

Thus of the total $7,845,679 paid on these claims, 62%, $4,857,146 went to attorneys and the cost of the system.

The object of this proposal is to achieve reallocation of those dollars so that the claimant will receive more of the total dollars spent, and the system will receive less. This can be achieved through a modified no-fault system patterned somewhat after the Workers' Compensation laws, which this proposal calls "The Patients' Compensation Plan" (5). The basic tenets of this plan are these:

1. Those who are injured in using the health care system should have a source to recover reasonable compensation for those injuries, regardless of fault.

2. Such compensation should be paid by the health care provider in some instances and should be borne by the patient in some instances, the test being:

a. If the injury is the result of an inherent risk, previously identified and made known, of the health care procedure being followed, then the economic burden falls on the patient who will have access to health and accident insurance to cover the risk.

b. If the injury is due to some other cause within the health care delivery system, either negligence or unknown cause, then the economic burden falls on the health care provider, who will have access to liability insurance to cover the risk.

3. The procedure for determining reasonable compensation to be paid by the health care provider is a procedure similar to Workers' Compensation whereby medical costs and lost income are dealt with on an actual, as occurring basis, and the claimants legal fees are regulated.

4. Cases in dispute would be heard by a hearing officer as in Workers' Compensation and decided by a Patient's Compensation Board similar to the Worker's Compensation Board, with two major additions:

a. The Patient's Compensation Board would have an advisory board of medical specialists to render opinions on "inherent risk" questions and technical medical questions.

b. The Patient's Compensation Board would have, as part of its operations, procedures for peer review of all medical procedures involved in claims and prompt referral to licensure boards or regulatory bodies of incidences of apparent deviation from a reasonable standard of care.

5. Each patient would have the opportunity to elect not to be covered under the system, just as each worker can elect not to be covered by Workers' Compensation, in which case a claim for an injury would be dealt with in the existing court system.

6. The plan would cover all aspects of the health care system and all the licensed professionals and institutions.

7. Some of the details of the procedures are as follows:

a. Each patient entering the health care delivery system would come under the plan, unless he elected in writing not to. Each health care professional and institution would be required to come under the plan.

b. The "inherent risk" would have to be determined in advance and specified in writing, as is currently done under the doctrine of "informed consent." These determinations would be made by the health care professional with the assistance of his specialty society or the Advisory board to the Patients' Compensation Board.

c. When a compensable incident occurs, the patient must give prompt notice, and a claim must be brought within one year of the occurrence, with a five-year cap for discovery, and by a minor under six, by his eighth birthday.

d. Compensation will be paid in the form of all necessary medical attention and lost wages under a formula similar to the Workers' Compensation formula. When the injured party is a non-wage earner, compensation can be paid for lost services or for future lost wages.

e. Compensation can be offset by other collateral sources of payment available to the patient, less the cost of obtaining such collateral source. Compensation is limited to economic losses.

f. Rehabilitation is a major part of the recovery benefits, as it is in Workers' Compensation.

g. Death is a compensable event only when there has been negligence as a causative factor.

h. Special provisions are made for emergency treatment where inherent risk can't be communicated and where there is no real opportunity for a patient to elect to come under the act.

i. Compensation can be increased where there is serious fault and decreased when the patient does not follow instructions.

j. Continuing physical exams of claimant can be required, and medical and hospital bills must be reasonable and are under the control of the Board.

k. Cases may be subsequently reopened where the disability has improved or worsened.

l. Attorney's fees for the claimant are limited to 20% of the first $25,000 of recovery, 15% of the next $10,000 and in no case to exceed $6,500. These fee limitations are identical to the current Workers' Compensation act in Kentucky.

m. The Patients' Compensation Board of 15 members are split with five members from the health care delivery system, five from the legal profession, and five from the public.

n. The Board would have hearing officers who would gather the facts and make recommendations, as in Workers' Compensation.

o. The Board would receive administrative services from the State Department for Human Resources, or a like governmental Agency.

p. The Advisory Committee to the Board would be all health care professionals with access to Ad Hoc Committees from all the specialties and institutions.

q. There would be continuing peer review by the reporting of incidents to the Board, and the review of such incidents to the Board, and the review of such incidents by the Advisory Committee with referral to the licensuring boards where appropriate.

r. Patients coming under Federal Tort Law would be exempt from this act.

s. No cases would go to court for a jury trial. There would be limited appeals from Board decisions to the Circuit Court.

t. Insurance will be available to health care providers, who may also become self-insured or pool their risk as currently in Workers' Compensation.

u. Insurance will be available to patients to cover inherent risks, and would be health and accident insurance policies somewhat similar to "trip insurance."

v. There are penalties for bringing claims without reasonable grounds.

The major changes that this proposal will bring over the current tort system are set out below, with some comments pro and con on their anticipated effects.

a. Everyone who is injured in the system would be compensated without regard to fault, except in the case of death, or when the injury is an inherent risk.

Many observers believe that a "no-fault" system would bring to the surface many injuries that are occurring in the health care system and are not being prosecuted now because of the cost and difficulty involved. They believe that even through the proposed system might be less expensive on the known claims, the "unknown" claims that would come to light would cause expenses to soar (6).

My response is that there probably are a lot of injuries that the proposed system would uncover—but if they are occurring why shouldn't they come to light? To the extent that these new claims are exposed, economic pressures would work towards eliminating the causes. The costs of these injuries are now being borne by some segment of society and the more we know about them the better we could deal with them.

Death is excluded from the no-fault system because almost all deaths take place within the system and can't be compensable unless there was negligence in causing the death.

b. All injuries would be reported to a responsible body for peer review and appropriate steps could be taken to eliminate the causes.

Currently a negligent act in the delivery of health care can result in monetary damages paid by an insurance company, but little else happens. Under Kentucky law the insurance companies report all claims payments to the Department of Insurance which in turn advises the Kentucky State Board of Medical Licensure of the name of the doctor and the amount of the settlement, and if a doctor gets enough paid claims against him the Medical Licensure Board will look into it. This is far short of an efficient on-going process of review of the quality of medical care. This proposal provides the mechanism and makes it mandatory, that continuing peer review of quality of care be performed, and ties the system directly into the responsible licensing authorities and brings the scrutiny not only of doctors, but on all providers in the health care delivery system.

Critics will most likely say we will be creating another bureaucracy that will interfere and hassle the health care system, and make it more expensive. In my opinion the review system would have to be well run, or the critics could be right.

c. The identification of inherent risks and the elimination of compensation for such risk.

The technological and scientific developments in diagnostic techniques and treatment and the invasiveness of many of these, and the development and use of new drugs, all lead to increasing risks in our health care system. This proposal requires that these inherent risks be determined and disclosed in writing and in advance to the patient. This is currently being done (or should be done) to satisfy the requirements of "informed consent." There is often the question of "how great is the risk" and might the patient be better off not knowing some of the risks. This proposal is in favor of disclosure and the patient knowing the risks. It is believed that the pressures to determine and disclose risks in order to avoid possible liability, in conflict with the often existing pressures to proceed with treatment and minimize any real analysis of risks, will result in an accommodation where risk will be determined and disclosed in a reasonable manner. There is also, as part of this proposal, an Advisory Board to the Patients' Compensation Board made up of medical expert professionals who will assist in determining inherent risks and in settling disputes concerning them.

There are conflicting views on the treatment of risks in modern medicine and some of those concerned with hospital occupancy and expensive procedures may feel that the disclosure requirements are too stringent, and not necessary, and not in the patient's best interest. The author of the proposal believes otherwise.

Where injuries occur because of an inherent risk in any procedure, there will be made available accident and health insurance applicable to these inherent risks that an individual is assuming, and this insurance will be available like "trip insurance" for a premium to each patient as he enters the system. This "trip insurance" will be offered by private insurance carriers (7).

d. The proposal would change the current statute of limitations applied to minors.

The proposal adopts the existing statute of limitations in Kentucky on medical malpractice claims for adults of one year (with five years for discovery), but changes the rule as to minors. Currently no limitations run against minors until they reach their 18th birthday, and the proposal changes this so that the statute starts to run at age six. This means that a parent or guardian would have to assert a claim for a minor over six year of age in the same way a claim has to be made by an adult.

Critics will assert that this is unfair to impose on one who cannot fend for himself and may later disagree with a parent or guardian's decision on prosecuting a claim. I believe, in fact, almost all serious claims involving minors are brought promptly by the parent because of the economic necessities, and leaving the door open for a claim for as much as 23 years brings unfair uncertainty and exposure to pediatricians and obstetricians. Many states have adopted similar statute of limitations for minors (8).

e. The proposal will do away with jury trials.

There will no longer be any necessity for a jury trial to determine fault. The questions will be: was there any injury; was it caused by the health care system; was it an inherent risk. If the answer to the first two questions is yes, and to the third question is no, then an award must be determined. All of these decisions will be made by an Administrative Board in informal procedures with very little expense.

If the concept of no fault is accepted then there can be little criticism of eliminating jury trials. They are not needed. But critics will object as the right to a jury trial lies deep in our anglo-saxon experience over some 800 years. It has been our major civil system for holding people accountable for their acts.

It is the foundation of this proposal that jury determination of fault in a medical malpractice case is so expensive we can no longer afford it, and therefore we do away with fault in awarding compensation and substitute a more efficient and professional system of accountability through peer review of all accidents and claims. We eliminate the expenses, but maintain the deterrence.

Critics will also question the creation of another Board, another government bureaucracy, another political entity and the further intrusion of government in the private practice of medicine. These are serious concerns. But the bottom line is the private practice of medicine is already a public matter and the cost of medical malpractice is a public concern and the purpose of government is to deal effectively with social problems. We have a social problem and if government is further involved we must see to it that it is done effectively.

f. Attorney fees are regulated.

The proposal adopts the regulation of claimant's attorney fees that is now the law in Kentucky in Workers' Compensation cases — 15% to 20% of the award, not to exceed $6,500 in any case. This will reduce these costs to claimants, on average, by over 60% (9).

Additionally, the defense attorney costs on a contested case in Workers' Compensation average between $2,250 and $2,500. This reduction over current defense costs will also average over 60%. These savings, along with savings on expensive expert witnesses, and the recoveries from collateral source prepaid medical insurance are the economic basis that will make this proposal work.

g. The proposed compensation benefits are limited, exclude any award for pain and suffering, and the plan takes advantage of some collateral sources available for reimbursement.

The proposal has adopted the payment schedule currently in effect in Kentucky for Workers' Compensation which pays all medical expenses, actual lost wages and a cap equal to the average wage in Kentucky, and no award for pain and suffering.

Critics may assert that the limitations are arbitrary and are unfair to the injured. They could be right, it depends upon your perspective and beliefs concerning the allocation of resources. The system was chosen because it actually works fairly well in Workers' Compensation cases in Kentucky and it does cover all, or the major part, of the true economic loss, and it has the flexibility to be increased of decreased according to the future needs of the injured party. It there are adequate resources available the structure of the awards could be increased and awards of pain and suffering could be included. It is my belief that this should not be considered until some experience has been achieved with the plan in effect.

In many of our states, including Kentucky, there are serious problems over the increasing costs of long standing Workers' Compensation systems and the question logically arises whether the proposed Patients' Compensation system will inherit these cost problems. The answer initially at least, is no. In Kentucky, for instance, the major increases in costs have developed from the Special Fund and the Kentucky Reinsurance Association; neither of which are involved in the traditional Workers' Compensation insurance system, and neither will exist under the Patients' Compensation system.

It is true, however, the largess of a state legislature or of the courts can subsequently effect any system of compensation for injury, but the specific terms of this Patients' Compensation proposal will hold costs to reasonable and predictable levels. These costs are projected in detail in the next section of this paper.

h. Comparative cost analysis.

A group of 100 closed claims handled by the Kentucky Medical Insurance Company under the existing tort system were selected for a cost analysis to compare the actual amount and distribution of payments under the tort system with the anticipated payments and distributions under the proposed Patients' Compensation system. This group of claims are from the same 30-month study group referred to earlier in this paper, but were selected from the most recent period to more adequately reflect our current results (10).

These closed claims run the gamut from the very serious to the very trivial: some were settled, some went to trial, and some were dismissed or abandoned. We know the actual costs under the tort system and we have determined the probable costs under the Patients' Compensation system.

These results clearly disclose the waste of economic resources in trying to determine fault under the current tort system, and how a Patients' Compensation system, properly administered can produce better net benefits to the injured parties for about half the cost.

This comparative cost analysis is set forth in Table A.

I envision a system where an individual entering any health care facility would become a member of the Patients' Compensation plan and would be informed of the inherent risks, if any, of the treatment he is to receive. He will have available to him health and accident insurance to cover those inherent risks. He will further have the Patients' Compensation plan to provide benefits if he is injured by negligence or unknown cause. In the event of such injury, he or his family would fill out a simple claim form which would be processed by the health care facility or its insurance carrier. A prompt determination would be made if this was an injury which was not an inherent risk, and a prompt determination of benefits for lost income and medical expenses would be made and would be paid. If there is evidence of negligence by any health care provider, the claim will be referred by the Patients' Compensation Board to its medical specialist Advisory Board which would review the facts of the claim and if necessary refer the matter on to the appropriate licensing authority for peer review and remedial action.

Absent from this system are the costs and delays that

Table A
Comparison of Payments and Distribution
100 Closed Files

Tort System		Patients' Compensation System	
Total Paid	8,064,156	Total Paid	4,358,232
Indemnity Payments	7,170,099	Indemnity Payments[12]	4,165,732
To Claimant	3,585,050	To Claimant[13]	3,938,473
To Claimant's Attorney[4]	2,868,049	To Claimants' Attorney[14]	180,759
To Costs[4]	717,000	To Costs[14]	46,500
	7,170,099		4,165,732
To Defendant's Attorney and Costs[11]	894,057	To Defendant's Attorney and Costs[15]	192,500
	8,064,156		4,358,232
Total Insurance Costs[16]	**8,064,156**	**Total Insurance Costs**	**4,358,232**
Net to Claimants	**3,585,050**	**Net to Claimants**	**3,938,473**

have become the most prominent feature of determining fault under our current Court system.

What is proposed is that one or more states establish a five-year experimental program to test the fairness and efficiency of the proposed Patients Compensation system; enact the legislation which is detailed in this proposal with a five-year sunset provision and establish methods for monitoring the effects on those injured, as compared to the general results currently under the tort system, and monitoring the cost to the insurance carriers offering the Patients' Compensation coverage (17). If the studies done thus far are correct there should be a substantial reduction in cost without a concomitant net loss to those injured in the health care system.

NOTES

1. *Business Insurance,* August 18, 1986, presents the most recent compilation of State Legislative actions during 1986 in tort reform. More than 16 states have adopted some cap on non-economic damages, 13 states have statutes allowing for periodic payments, 6 states regulate attorneys contingent fees, and 11 states have recently enacted statutes allowing for credit for collateral source payments. A number of other state legislatures enacted similar legislation during the medical malpractice crisis in the mid-seventies.

In Congress, the *Moore-Gephardt Bill* (H.R. 3084, the "Medical Offer and Recovery Act") is pending before the Ways and Means Committee, and has been under consideration for the last several years. This proposal is to encourage prompt offers of settlement in medical malpractice cases and to limit recoveries to economic damages. The Bill contains an approach recommended by Professor Jeffrey O'Connell of the University of Virginia Law School. See: H. Moore, J. O'Connell. "Foreclosing Medical Malpractice Claims by Prompt Tender of Economic Loss." Louisiana Law Review, Volume 44 #5, May 1984.

There has also been introduced in Congress the "Federal Tort Claim Reform Act of 1986," the "Government Contractor Liability Reform Act of 1986," and the "Product Liability Reform Act of 1986," all supported by the Administration and dealing with many of these same proposed remedies.

2. The author, Carl L. Wedekind, Jr., is an attorney (University of Virginia. 1950) who was in the private practice of law (Stites and Harbison) in Louisville, Kentucky from 1954 to 1981. He specialized in civil law including tort litigation (primarily, but not exclusively, for Defendants) and general corporate and insurance law. As attorney for the Kentucky Medical Association he set up a physician-owned medical malpractice insurance company (the Kentucky Medical Insurance Company) on behalf of Kentucky doctors and served as General Counsel and Director (1978–1980) and in 1981 became President and Chief Executive Officer of Kentucky Medical Insurance Company, in which capacity he still serves.

The author has been aided by too many people and their individual ideas to attempt to list, with the exception of two: Mr. Glen Schilling, a prominent Louisville attorney and past-president of International Association of Industrial Accident Boards and Commission, and a member of the Kentucky Workmen's Compensation Board, who provided technical expertise in regard to Workmen's Compensation law: and Mr. Larry Hamfeldt, also a prominent Workmens' Compensation attorney, who assisted in doing the economic closed claims study in converting the costs of cases under the tort system to the proposed Patients' Compensation system.

3. The Physicians Insurers Association of American (PIAA) compiles data from all the physician owned medical malpractice carriers, and the consistency of the data can be seen in their periodic reports. The most recent is the "1985 PIAA Data Sharing Reports," covering 4,760 closed medical malpractice files.

4. The Plaintiff's attorney's contingent fee and costs is an estimate based on informal polls conducted by the KMIC claims department personnel. The amount will vary depending on rural-urban considerations, and the seriousness and difficulty of the case. The average contingent fee selected is 40% of the gross recovery with additional expenses estimated at 10%.

5. A Workmens' Compensation type approach is under consideration in a number of forums. In Missouri, Representative Banton introduced H.B. 1628, "Medical Injury Compensation Law" which limits recovery to economic losses but does require the proof of negligence. No reported action was taken in the Legislature on this bill.

In Michigan, during the 1977 Legislature, a bill was introduced for the establishment of a State Medical Compensation Board, but the bill was not passed.

In Florida, State Senator Dempsey Barron introduced. "An Act Relating to Medical Incident Compensation," which would remove malpractice disputes from tort law and place them in equity under contract law and thus eliminate a jury trial. The 1986 Legislature failed to act on this bill.

The Workmans' Compensation approach has also been supported by a Tallahassee, Florida attorney, Frederick B. Karl, and a Wharton School of Finance professor, Patricia M. Danzon.

There may well be others that have not come to the author's attention.

6. The "Alliance of American Insurers" for instance established the Alliance Medical Malpractice Task Force which reviewed the issue of Workmens' Compensation type schedules for malpractice claims. Mr. John P. Waligore, attorney for the Alliance, reported to my

colleague, Glen Schilling (by letter of May 22, 1986), "Our Task Force rejected this approach because we viewed it as inappropriate at both the state and federal levels. We felt that such a system would not cut total claim costs because it could increase the frequency of claims brought significantly. Such a system would make claims much easier to bring and would make the certainty of an award much greater. The Task Force believed that a Workers' Compensation type system would probably increase total claim costs because claims that would formerly have been of too small value to merit filing an action in court and not be brought administratively. The Alliance does not recommend support for such a system.

7. The Kentucky Medical Insurance Company is prepared, subject to the approval of the appropriate State Department of Insurance, to offer this coverage.

8. Alabama provides a minor under four has until his eighth birthday; Indiana provides a minor under six has until his eighth birthday, and the two year statute applies thereafter. The courts in other states, such as New Hampshire, Ohio and Texas have declared provisions similar to these to be unconstitutional under their state constitutions.

9. The current average payment per indemnity case on closed files studied by the Kentucky Medical Insurance Company is $53,000, of which it is estimated 40%, $21,200, goes to the attorney. Thus the proposal would reduce the cost to a maximum of $6,500, a savings of at least $14,700.

10. After the exclusion of duplicate files where there was only one incident but a number of defendants, and the exclusion of some of the trivial claims where there would be no award or significant expense under either system, the remaining files compose the 93 included in the study.

11. Defendant's Attorney fees and costs are actual payments made in the 100 closed files.

12. Indemnity under the proposed Patients' Compensation plan is a combination of lost earnings, past medical expenses incurred and estimated future medical expenses. For the 100 cases, the total past and estimated future lost wages were computed on the Kentucky Workmen's Compensation formula and discounted to a lump sum payment, and totaled $3,311,996. The medical payments totaled to $2,125,909 before recovery of health insurance reimbursement, which was $1,272,172 leaving net medical payments of $853,737.

Industry estimates in Kentucky are that 85% of the population has some type of prepaid health insurance covering an average of 75% of total costs, .85 x .75 = .64. From this recovery we deduct the estimated premium paid for the health insurance ($75.00 per month) for a 2-year period, $1,800 per case. This computation results in the net medical payout of $853,737. Thus, the total indemnity of lost wages and past and future medical expenses totals $4,165,732 for the 100 cases.

13. This is net to claimant after deducting estimated costs and attorney fees.

14. Claimants' costs for attorneys in contested claims are regulated by the proposal in the same way they are regulated in Workmens' Compensation cases — a contingency fee limited to 20% of the first $25,000 lost wages recovered, 15% of the next $10,000, with a $6,500 cap. On the 100 closed claims studied, there were 19 cases where the full $6,500 fee would be earned, for a total of $123,500 and the total of the remaining awards produced an additional $57,259 in attorney fees, for a total of $180,759.

The additional costs for presenting the claims is estimated at $1,500 per case times an assumed 33% of the cases (31) .31 x 1,500 = $46,500.

15. The Defendant's costs for attorney's in contested Workmens' Compensation cases in Kentucky are not regulated and it is estimated in the industry that the average costs to defend these cases is between $2,250 and $2,500. We selected $2,500 as the average amount to be used and applied it to every closed claim in which we had paid defense costs. This was 77 out of the 100 files for a total cost to the defendant of $192,500.

16. These costs include payments made by all defendants in these closed claims, and thus are significantly higher than the earlier study which only included KMIC's costs.

17. The Kentucky Medical Insurance Company will be prepared to offer this coverage, subject to approval of the appropriate State Department of Insurance, on a trial retrospective premium basis.

An Open Letter

From the desk of Dr. Charles W. Townsend

Medical Director, Alaska Autologous Blood Ban

I would like to introduce you to an expanded blood service that will be available to you and your patients in January 1988 through Alaska Autologous Blood Bank.

The increased interest in the value of autologous transfusions has led all of the major blood suppliers n the nation and the American Medical Association to endorse the use of autologous blood whenever possible.

Autologous blood products can be provided by several mechanisms. The first and the most common is the use of predeposit programs where patients store blood in a iquid state for an anticipated need n the immediate future. Donations of blood can begin 35 days prior to the anticipated date it will be required and continue up to 72 hours prior to the needed time. These programs are in effect nationwide and represent the most common option employed when autologous blood is requested. For many situations this represents the most cost- effective mechanism available.

A second option to supply autologous blood is the use of Intra-operative Autotransfusion where blood shed during surgery is recycled during the surgical procedure and returned to the patient.

LONG-TERM FROZEN STORAGE: A third option is use of autologous units deposited by an individual and stored in the frozen state for use at a later time.

Alaska Autologous Blood Bank is now offering such a storage program as an option for your patients.

While blood collected and stored in the liquid state can be maintained for 35 days, frozen blood can be maintained for seven years under current AABB standards. Outlined below are some of the reasons for considering autologous transfusions.

AUTOLOGOUS TRANSFUSION ADVANTAGES :

1. Compatibility is assured and there is no risk of a transfusion reaction from the blood itself.
2. If the blood is properly processed there is no risk of disease transmission. Although AIDS has been the major concern of the population, a more significant consideration is Non A Non B Hepatitis which is a complication in 8-12 % of transfusions. Patients who develop Non A Non B Hepatitis are at significant risk of progressing to chronic active hepatitis. HTLV 1 which causes T cell leukemia can be transmitted by blood as can the new LAV 2 virus which causes an AIDS-like illness which i not detected by present screening tests. Hepatitis B, CMV, and the E virus can also be transmitted by blood.
3. There is no risk of isosensitization when autologous units are used.

IMPROVED TECHNIQUES:

The U.S. Navy has developed techniques which allow for the long term storage and transportation o frozen blood. And it has developed closed system of blood collection which eliminates the risk of contamination. This system has only recently become available fo civilian use.

ADVANTAGES OF OUR PROGRAM:

1. Patients who have rare blood types, IgA deficiency and those wh are isosensitized by prior blood exposure will have a reliable blood supply available for them.
2. Patients who are willing to don blood for an elective surgical procedure but cannot tolerate frequent phlebotomies for medical reasons can still participate since the deposit process can be performed at a schedule safest fo the individual.
3. The program allows for greater flexibility in

gical scheduling since the units viable until thawed and there is fear of losing the blood if gery has to be postponed prior to unit(s) being thawed.

It allows individuals who wish to sure themselves of an autologous od supply at all times the portunity to do so.

It allows families to participate, suring each member of the family eliable and safe blood supply from ir relative, if the blood is npatible.

FORDABLE:

While the storage of blood is an pensive undertaking, Alaska ologous Blood Bank has made this cess affordable through a g-term storage program which ws for 5 units of a person's od to be stored for possible ure use, or for a prescribed mber of units to be stored for a nned medical procedure.

W THE SERVICE WORKS:

Alaska Autologous Blood Bank will swer any questions concerning the gram. Scheduling can be anged by the physician or the ient. When blood is collected m a physician- referred patient a specific surgical procedure, horization from the physician be required. Such horization will indicate the date surgery, the hospital, the number units needed, the health history he donor, and how low a matocrit the physician feels is e. The donation is arranged at a e convenient to the patient.

All information which is necessary to insure patient safety will be obtained and entered into a computerized inventory system. This information includes the patient's name, birthdate, social security number, and a special donor number which is attached to each unit, allowing for multiple checks to be made to ensure accuracy. In addition to this information a bar code is assigned and the patient's photograph is attached to each unit to make unit retrieval efficient and accurate.

All necessary testing is done to ensure that the unit of blood meets all applicable standards of the AABB. If the unit of blood does not meet the criteria of the AABB, it can still be reserved for autologous use exclusively after approval by the medical director of Alaska Autologous Blood Bank and/or the patient's physician.

Using the Navy's improved methodology, the blood is collected and separated into components; and the red cells and plasma (if needed) are then made ready for freezing at -80° Centigrade in specially designed freezers. The freezers are continuously monitored and back-up systems are available in case of a power failure. The freezers themselves are housed in secure, temperature- controlled buildings.

If the blood is needed it is the responsibility of the patients to notify their physician that they are participants in the program and to give instructions on obtaining their stored units. If the patient is not capable of communicating as a result of illness, a Medic Alert® wallet-size card and a bracelet or necklace I.D. on the patient will have all necessary information. The physician can arrange to have the units made available by notifying Alaska Autologous Blood Bank.

Prior to release of the units all identifying parameters will be checked including the bar code and the photograph taken at the time of donation. This will insure that the correct units are released. The blood is then thawed, washed, and made available for transfusion.

REDUCE RISK:

This program of liquid and frozen autologous blood will reduce both hospital and physician medical/legal risk and will result in a significant increase in the quality of care in our community.

Alaska Autologous Blood Bank, located at at **9500 Independence Blvd., Suite 300**, is proud to provide this service to Anchorage residents. Please feel free to call at **344-6669** any time for further information.

P.S. I wish this had been available when I had my heart transplant.

History of Medicine in Alaska

J. RAY LANGDON, M.D.

The importance of the late J. Ray Langdon, M.D., is the vitality and direction of the mental health program in Alaska today.

In a paper on the history of psychiatry in Alaska, Dr. Langdon wrote: "Prior to 1946, to my knowledge, no psychiatrist resided in the territory though some may have been stationed with the military during World War II. Thus, from the 1890s until mid-century the only treatment facility for Alaskans was Morningside Hospital in Portland. This hospital was founded in the 1880s as a private asylum, accepted occasional Alaskan patients after 1900, and became the only contractor from 1904 until 1961."

In 1957, one year before J. Ray Langdon came to the territory, there were two psychologists and two psychiatric social workers in Alaska: a pair in Juneau, another in Anchorage. One was James C. Parsons, still in private practice. The only psychiatrist in the territory was Virginia Wright (O'Malley) and as Dr. Langdon has stated, "As the mother of nine children, she was necessarily a part-time practitioner."

Dr. Langdon was Medical Director of Morningside Hospital where he was exposed to and became interested in the problems of mental health in Alaska. He was asked by the Commissioner of the Alaska Department of Health and Social Services to apply for a position in the Division of Mental Health. He became Assistant Director, living in Anchorage. His job was to plan for the development of mental health programs throughout the then anticipated State of Alaska and to oversee the planning of Alaska Psychiatric Institute (API). He came to Alaska in April 1958; statehood was passed by Congress in June.

After two years, disagreeing with the politicizing of mental health problems, he resigned from the post. Henry Coe, Director of Morningside Hospital, tried to persuade him to return to Portland.

Giving it considerable thought, J. Ray went into private practice. It was not his original intent to start a clinic, but as he slowly acquired personnel, he found himself Director of the Langdon Clinic which by 1971 embraced ten professionals plus reading specialists and later a drug staff. The Clinic was to contract with the federal government for a Narcotics Addicts Rehabilitation program.

In the meantime, Alaska developed three mental health systems which were located in the same geographic areas but did not serve the same patients. These were the state Division of Mental Health, the Public Health Service, and the military. Because Alaska could not easily attract psychiatrists to its largely rural situation, private psychiatrists served as consultants for all three. In addition, J. Ray was involved with the State Court System and the Division of Vocational Rehabilitation.

Also, he was instrumental in the passing of the Community Mental Health Center Act. This was implemented in 1974, establishing community centers, big and small, reaching from one end of the state to the other, including some native corporations. Its intent was to meet community mental health needs, with emphasis on those released from API who needed continued support.

The University of Washington began a medical program to serve northwestern states without medical schools, specifically Alaska, Montana and Idaho, known as WAMI. Dr. Langdon was clinical professor of psychiatry, teaching third and fourth year medical students and third year residents.

Finally, he pushed for an acute care unit at Providence Hospital which currently has eighteen beds, four for adolescents. These are for patients needing brief institutionalization for problems. It is here that a cheerful tapestry has been placed in his memory.

He was frustrated with the treatment of the mentally ill, especially those who were released from institutions which then abandoned them. Thirty-five percent of street people are probably in this group.

Always outspoken, he did not hesitate to attack issues be they of mental health, related subjects such as penal reform or abortion or ethical problems, drugs, the Alaska Native or women's liberation. He even prodded the medical profession, proposing "that the Alaska State Medical Association has the right and responsibility to be concerned and to voice opinions about many social problems which it has been reluctant to do."

It is not surprising that his public personality was controversial, but this is not the way his family and friends found him.

Joseph Raymond Langdon was born on July 8, 1924, in Pueblo, Colorado. He went to Colorado College and was to graduate from St. Louis University School of Medicine in 1950 but not without incident.

He was enrolled in an accelerated medical program and during a break from March to June had gone to work for the Colorado Fuel and Iron Corporation. Working in the laboratory, a leaking bucket of creosote was ignited by a bunsen burner and burned J. Ray severely on the chest, face, neck, and right arm. In those days, the standard treatment for burns was sulfa and paraffin spray with the patient under a heat crib. The removal of the paraffin was an exceedingly painful process. His nurse throughout this ordeal was Thelma Perse.

J. Ray recovered and married Thelma.

She says about him that he was a man with no hobbies or outdoor interests. He did volunteer for Little League and became greatly involved. His pleasures were politics and his practice. He was curious about "what is going on today?" and how it related to history. He was easy to have about, seldom angry, with a good sense of humor. He was encouraging to Thelma and proud of her accomplishments.

She was appointed by Governor Egan to the Office of Child Advocacy which lasted for about five years and then by Governor Hammond to the State Board of Education, a marvelous experience.

With both the Langdons it would be too easy to make lists of their many commitments, accomplishments and awards. Alaska has been fortunate to be so well served.

The Langdons have five children and three grandchildren. One of them remarked to Thelma after J. Ray's death on September 3, 1981 that "There are so many questions I have to ask Dad."

Gwynneth Gminder Wilson
Chairman, ASMA Auxiliary History Project

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AIR FORCE

Glimpses of Alaskan Medical History

Edited by Robert Fortuine, M.D.

A frustrated doctor at the Shumagin Islands (1741)

The name of Georg Wilhelm Steller is known to every Alaskan for its association with several birds, sea mammals, plants, and natural features that he was the first to describe, or that were named in his honor. As the naturalist and surgeon on Vitus Bering's ship *St. Peter,* Steller had the unusual opportunity, in 1741, to be the first to view a pristine land with the eye of a scientist. Unfortunately, the time available for study and exploration was minimal.

Throughout his journal, which was obviously rewritten later from his daily notes, Steller displays bitterness because his knowledge and advice were repeatedly ignored, often to the detriment of the crew's welfare. There seems little doubt that several of the officers and men resented Steller's presence on the ship, possibly in part because they had never encountered a scholar before, least of all one like him. In the passage quoted, the doctor's rancor constantly breaks through, leading to his most unworthy resolve to forget the welfare of the rest and henceforth look out only for himself. He did not carry out his threat, of course, and in fact made himself indispensable to the survival of many during the coming winter of shipwreck on Bering Island.

The text presented is based on Steller's diary for August 30 and 31, 1741, while the ship was anchored in the Shumagin Islands to take on water. Although he would have preferred to spend his limited time exploring the natural history of the islands and studying their people, his first concerns were fresh water and antiscorbutic herbs for the long voyage home. Steller received little encouragement in his quest, and ultimately the ship's company paid a heavy price for not heeding him. Thirty-two men had perished from scurvy before the stragglers reached home a year later.

— — — — — — — —

"Aside from the red whortleberries and the black crowberry, or *shiksha (Empetrum),* which are found here in the greatest abundance, the products of the vegetable kingdom of greatest use to us were the glorious antiscorbutic herbs, such as *Cochlearia, Lapathum folio cubatali, Gentiana* and other cresslike herbs, which I gathered solely for my use and that of the Captain Commander. For although I had made representations that our medicine chest, from the very beginning, had been miserably supplied, inasmuch as it was mostly filled with plasters, ointments, oils, and other surgical remedies enough for four to five hundred men in case of a battle but had none whatever of the medicines most needed on sea voyages and serviceable against scurvy and asthma, our commonest cases; and although I had therefore requested the detail of several men for the purpose of collecting such quantity of antiscorbutic herbs as would be enough for all, nevertheless even this proposition, so valuable to all and for which I merited gratitude besides, was spurned. Later, however, there were regrets enough, and when we had scarcely more than four able-bodied men left on the vessel, I was tearfully begged to help and assist, which then, though with empty hands, I did to the utmost of my strength and means, although it was not my office and my services had always been scorned before the disaster. It must also at last have caused even the coarsest and most ungrateful persons to take notice when the Captain Commander, who from scurvy and confinement had entirely lost the use of his limbs, was restored by me to such an extent simply by his partaking of the fresh spoonwort that within eight days he could get out of bed and on deck again and felt as well as at the beginning of the voyage, and also when the use of *Lapathum* for only three days, according to my direction, made the teeth of most of the sailors firm again."

"Because of the rain which began towards evening I had already built myself a hut and intended to spend the night on the island; I decided nevertheless finally to return to the vessel in order once more to present emphatically and with the greatest respect my opinion on the bad water and on the collecting of the herbs. However, when I saw my opinion concerning the water again spurned and coarsely contradicted and had to hear myself, like a surgeon's apprentice belonging to the command, ordered to gather the herbs, and that this important work, which affected the health and lives of all, was not considered worth the labor of a few sailors, I repented of my good intentions and resolved that in the future I would only look after the preservation of my own self without wasting another word. With this in mind I went ashore again on the morning of August 31, continued my work, and together with Mr. Plenisner explored the land. . . . On this day we buried the first of our crew, the sailor Shumagin, who had died on the previous day almost as soon as he got ashore. The island is since called Shumagin's Island after him."

REFERENCE

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News from the American Society for Circumpolar Health

Arctic Atmospheric Pollution

International Conference Held in Cambridge, England

An international conference on Arctic Atmospheric Pollution was held at the Scott Polar Research Institute in Cambridge, England on September 2–5, 1985. The conference, the fourth since arctic haze was first observed and defined in 1956, was sponsored by the State of Alaska through the University of Alaska–Fairbanks and cosponsored by the Comite Arctique International, the United Nations Environmental Programme, the World Meteorological Organization, and the World Health Organization. It attracted approximately 40 internationally known scientists from ten countries, including five Polar Rim nations.

While the conference focused on arctic science issues of international concern, a more immediate benefit to Alaskans lay in its addressing public health concerns specific to air pollution.

The conference's third session, chaired by Dr. John Middaugh, who chaired the National Arctic Health Science Policy Task Force of the American Public Health Association, featured the following participants: M. Henderson (National Academy of Science, USA), "The epidemiology of health effects of air pollutants"; A. C. Upton (New York University), "Biological effects of low level ionizing and non–ionizing radiation"; W. C. Hanson (USA), "Ecological mechanisms involved in cycling of radionuclides in Arctic ecosystems"; C. Stutzman and D.M. Nelson (Centers for Disease Control, USA), "Cancer risk in Alaska natives due to fallout from atmospheric nuclear weapons testing"; W. Turner (Harvard University), "Indoor air pollution: a public health perspective"; J. C. Hansen (Hygiejnisk Institut, Denmark), "Actual exposure to heavy metal in Greenland: natural and man–made sources"; J. Harte, (University of California at Berkeley), "Ecological implications of Arctic haze."

One of the panel's concerns involved Alaskans exposed to radionuclide fallout following the atmospheric testing of nuclear weapons during the 1950s and 1960s. Problems related to radionuclide deposition in the Arctic were well-documented by Dr. W.C. Hanson, whose work demonstrated how pollutants transported to the Arctic can bioaccumulate to pose potential health threats.

The panel concluded that such testing had ceased before Alaskans suffered measurable health effects. After studying Hanson's data, they concluded that while the risks cannot be said to be zero, the number of cancer cases, the number of exposed individuals, and the radionuclide dose to exposed individuals were so small as to constitute no basis for public health fears. And, in this case, they stated, enough time had elapsed for cancers to develop in affected populations.

They agreed, however, that environmental degradation is occurring in the Arctic because of atmospheric pollution, and an assessment of the long-term, cumulative effects of the degradation on both health and the environment "cries for attention." Monitoring of the specific pollutants transported to the Arctic and measuring the extent of their deposition are necessary. Also needed, they stated, is research to trace the ways pollutants find their way through the ecosystem eventually to affect human health.

According to the panel, the impact arctic haze has on the ecosystem may well be a more sensitive indicator of change than its impact on human health. Any environmental impact may well affect human health far into the future, they noted, adding that because arctic populations are stressed, episodes of pollution may well affect them differently from other populations.

Thus monitoring of arctic pollution and conducting ongoing studies of its ecological effects to establish baselines and determine trends over time become critical public health concerns. A most critical need for the monitoring system is a statewide cancer registry, they argued.

Interestingly, the panel argued that urban and indoor air pollution and the dynamics between them constitute a more immediate health threat than arctic haze. The panel pointed out that urban residents in the Arctic spend approximately ninety percent of their time indoors during the long winters when arctic haze is prevalent. Threats to human health from urban atmospheric pollutants are not well–documented, they noted, citing the need for simultaneous indoor/outdoor air measurements to establish if measurable levels of auto emissions somehow find their way indoors.

Among the more important indoor air contaminants identified as being associated with health or irritation are passive tobacco smoke, radon decay products, carbon monoxide, nitrogen dioxide, formaldehyde, asbestos fibers, and microorganisms. Using unvented combustion appliances like kerosene heaters and remaining indoors for long periods in inadequately ventilated buildings were identified as having very high potential for adverse health effects.

At the request of the Alaska representatives, the conference participants tailored one recommendation specifically for the state, calling for it to develop and conduct a research and monitoring program to be used as a model for other Polar Rim nations.

Such a recommendation poses a challenge for the state, but it provides also an exciting opportunity. Until recently, arctic research has lacked a coherent national policy. It has been conducted by various federal, state, and private agencies with differing agendas. Now, with passage of the Arctic Research and Policy Act of 1984, the nation is moving towards establishing an integrated arctic research policy.

The proceedings of the 1985 Arctic Atmospheric Pollution Conference have been published. Edited by B. Stonehouse, Arctic Air Pollution is available from Cambridge University Press.

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News from the American Society for Circumpolar Health

WHO Study Group Focuses on Tobacco Use as the Most Important Cause of Cancer in Indigenous Northern Populations

by Anne P. Lanier, M.D., M.P.H.[1]

The following consensus statement was formulated during the recent 7th International Congress on Circumpolar Health in Umea, Sweden, June 8–12, 1987.

> **The WHO Study Group on Circumpolar Cancer strongly emphasizes the most urgent need to reduce or totally eliminate the use of all tobacco products. Lung cancer is on an alarming rise in indigenous circumpolar populations. Since the 1950s, lung cancer in women has increased 15 times in parts of the Arctic to a world record level and continues to rise due to cigarette smoking. A virtual epidemic of lung cancer is occurring and will continue in the coming generations without immediate reduction of use of tobacco products. Such a reduction would result also in decreases in other tobacco-associated cancers, heart and respiratory diseases, and low birth-weight babies.**

Following the 5th International Congress on Circumpolar Health in 1981 in Copenhagen, Denmark, a WHO Study Group on Circumpolar Cancer was established. The objectives were:

- to obtain accurate information on the incidence of cancer in all northern populations;
- to collect cancer data in a standardized way in order to allow accurate comparisons of data between populations;
- to identify areas for productive collaborative cancer research in the Arctic, and
- to share information on prevention and control programs.

The study group has met at each International Congress and annually between Congresses. Cancer patterns in indigenous Arctic populations differ from those of the majority population in each respective country. Some rare cancers occur at very high rates, including cancer of the nasopharynx, salivary glands, liver, and esophagus. Although these usually rare cancers appear at excess rates, cancers which are frequently seen in "Western" countries are now among the most common in the Arctic groups: colorectal, breast, lung, and stomach cancer. Data from Greenland, Alaska, and Canada presented at both the scientific session and cancer study group meeting at the recent 1987 Circumpolar Health Congress emphasized that lung cancer was among the most frequently diagnosed cancer in Native populations, and rates for lung cancer were increasing more rapidly than other sites. Data also verified high rates of tobacco use across the Arctic. As emphasized by the consensus statement, the greatest impact on the cancer problem among Native populations of the Arctic will come from programs for prevention and control of tobacco use.

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[1] Arctic Investigations Laboratory, Centers for Disease Control, 225 Eagle Street, Anchorage, Alaska 99501–2692

News from the Alaska Public Health Association

Opposition to Plutonium Shipments Under a Proposed United States Agreement with Japan

The American Public Health Association,

Noting that plutonium oxide powder is an extremely toxic, easily airborne nuclear fuel that can be lethal in minute amounts when ingested; and

Recognizing that the United States and Japan have recently concluded the negotiation of a 30-year nuclear cooperation agreement that would permit the shipping of plutonium by air over a polar route between Europe and Japan (1); and

Noting that the signing of the agreement would substitute a blanket 30-year approval for the present case-by-case review process that gives the United States ongoing control over the safety and security of plutonium shipments; and

Recognizing further that it is very likely that the agreement would result in the designation of an Alaskan airport as an intermediate refueling point for the shipments from Europe, across Canada and/or the United States to Japan; and

Noting that it is predicted as much as 40 metric tons of plutonium oxide powder would be shipped from Europe to Japan every two weeks for several years starting in the early 1990's; and

Noting that the treaty requires only that International Atomic Energy Agency standards are to be used, and these standards require a cash test velocity of only 44 feet/second, while USNRC standards require a velocity of 422 feet/second (1); and

Noting that a cask for safely shipping large quantities of plutonium by air has not yet been designed, and it is unclear whether it is possible to build a cask that can survive an airplane crash and is large enough to ship the quantities of plutonium contemplated by the agreement; and

Noting an environmental impact statement has not been prepared for the proposed agreement, although there is court precedent for the proposition that the movement of nuclear fuel qualifies as a major federal action requiring preparation of an impact statement; and

Realizing that in the event of a terrorist attack, in-flight mechanical failure, in-flight collision, crash on landing or take-off, the public health threat to the people of the United States and other countries exposed to hundreds of pounds of liberated powdered plutonium oxide could be catastrophic; and

Noting that the suit filed by the state of Alaska against the federal government on October 1, 1987 indicating that the agreement is illegal because it fails to take proper safeguards to protect the public health and environment, on October 3, 1987 was denied by the federal judge who refused to put an immediate halt to the consideration by the Department of Energy (USDOE) of the treaty with Japan; and

Noting further that the USDOE hurriedly pushed this agreement through to President Reagan's desk precisely to circumnavigate the Alaskan suit; and

Recognizing that the nuclear cooperation agreement between Japan and the United States has been hurriedly given approval by the Department of Energy, and at this moment awaits the signature of President Reagan, and subsequently, approval by the Senate; therefore

1. Opposes ratification of the nuclear cooperation treaty between Japan and the United States;
2. Advises President Reagan and all United States Senators of this position immediately;
3. Advises the Canadian government and the Canadian Public Health Association of this position.

NOTE: This resolution was passed unanimously by the American Public Health Association at its annual meeting, New Orleans, October 22, 1987.

REFERENCES

1. Paul Leventhal *et al.* Air Transport of Plutonium Obtained by the Japanese from Nuclear Fuel Supplied by the United States, Nuclear Control Institute: Washington, DC, March 3, 1987; House Joint Resolution No. 35, in the Legislature of the State of Alaska, Fifteenth Legislature — First Session, Relating to the use of state airports for plutonium shipments under a proposed United States agreement with Japan.

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News from DISTA about a new dosage form of cephalexin

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- Backed by over 15 years of clinical experience
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NEW Keflet Tablets are available as:

250-mg Tablets

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Keflet is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-sensitive patients.

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Brief Summary. Consult the package literature for prescribin
information. Indications and Usage: Keflet® Tablets (cephalexin, D
are indicated for the treatment of the following infections when caused by
susceptible strains of the designated microorganisms

Respiratory tract infections caused by *Streptococcus pneumoniae* and
group A β-hemolytic streptococci (Penicillin is the usual drug of
choice in the treatment and prevention of streptococcal infections,
including the prophylaxis of rheumatic fever Keflet is generally effe
tive in the eradication of streptococci from the nasopharynx, howe
substantial data establishing the efficacy of Keflet in the subsequer
prevention of rheumatic fever are not available at present)

Otitis media due to *S pneumoniae, Haemophilus influenzae,* staphylo
cocci, streptococci, and *Neisseria catarrhalis*

Skin and skin structure infections caused by staphylococci and/or
streptococci

Bone infections caused by staphylococci and/or *Proteus mirabilis*

Genitourinary tract infections, including acute prostatitis, caused by
Escherichia coli, P mirabilis, and *Klebsiella* sp

Note—Culture and susceptibility tests should be initiated prior to anc
during therapy Renal function studies should be performed when indica

Contraindication: Keflet is contraindicated in patients with known alle
to the cephalosporin group of antibiotics

Warnings: BEFORE CEPHALEXIN THERAPY IS INSTITUTED, CAREFUL INQUIRY SH
MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORIN
PENICILLIN CEPHALOSPORIN C DERIVATIVES SHOULD BE GIVEN CAUTIOUSLY TO PE
SENSITIVE PATIENTS

SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND
EMERGENCY MEASURES

There is some clinical and laboratory evidence of partial cross-allerge
icity of the penicillins and the cephalosporins. Patients have been report
to have had severe reactions (including anaphylaxis) to both drugs.

Any patient who has demonstrated some form of allergy, particularly
drugs, should receive antibiotics cautiously No exception should be ma
with regard to Keflet.

Pseudomembranous colitis has been reported with virtually all broad-
spectrum antibiotics (including macrolides, semisynthetic penicillins, an
cephalosporins), therefore, it is important to consider its diagnosis in
patients who develop diarrhea in association with the use of antibiotics
Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of t
colon and may permit overgrowth of clostridia Studies indicate that a
toxin produced by *Clostridium difficile* is one primary cause of antibio
associated colitis

Mild cases of pseudomembranous colitis usually respond to drug di
continuance alone In moderate to severe cases, management should
include sigmoidoscopy, appropriate bacteriologic studies, and fluid, elec
trolyte, and protein supplementation. When the colitis does not improve
after the drug has been discontinued, or when it is severe, oral vancom
is the drug of choice for antibiotic-associated pseudomembranous coli
produced by *C difficile*. Other causes of colitis should be ruled out.

Usage in Pregnancy—Safety of this product for use during pregnan
not been established

Precautions: *General*—Patients should be followed carefully so that
side effects or unusual manifestations of drug idiosyncrasy may be det
If an allergic reaction to Keflet occurs, the drug should be discontinued
the patient treated with the usual agents (eg, epinephrine or other pres
amines, antihistamines, or corticosteroids).

Prolonged use of Keflet may result in the overgrowth of nonsusceptil
organisms. Careful observation of the patient is essential. If superinfect
occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment w
the cephalosporin antibiotics. In hematologic studies or in transfusion
cross-matching procedures when antiglobulin tests are performed on t
minor side or in Coombs' testing of newborns whose mothers have
received cephalosporin antibiotics before parturition, it should be reco
nized that a positive Coombs' test may be due to the drug.

Keflet should be administered with caution in the presence of marke
impaired renal function. Under such conditions, careful clinical observa
and laboratory studies should be made because safe dosage may be
than that usually recommended.

Indicated surgical procedures should be performed in conjunction w
antibiotic therapy

As a result of administration of Keflet, a false-positive reaction for gl
cose in the urine may occur. This has been observed with Benedict's
Fehling's solutions and also with Clinitest® tablets but not with Tes-Ta
(Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in in
uals with a history of gastrointestinal disease, particularly colitis.

Usage in Pregnancy—Pregnancy Category B—The daily oral adm
tion of cephalexin to rats in doses of 250 or 500 mg/kg prior to and du
pregnancy, or to rats and mice during the period of organogenesis onl
adverse effect on fertility, fetal viability, fetal weight, or litter size. Note
safety of cephalexin during pregnancy in humans has not been establ

Cephalexin showed no enhanced toxicity in weanling and newborn
as compared with adult animals. Nevertheless, because the studies in
humans cannot rule out the possibility of harm, Keflet should be use
pregnancy only if clearly needed.

Nursing Mothers—The excretion of cephalexin in the milk increase
4 hours after a 500-mg dose; the drug reached a maximum level of 4
then decreased gradually, and had disappeared 8 hours after administ
Caution should be exercised when Keflet is administered to a nursing

Adverse Reactions: *Gastrointestinal*—Symptoms of pseudomem
ous colitis may appear either during or after antibiotic treatment Nau
and vomiting have been reported rarely. The most frequent side effect
been diarrhea It was very rarely severe enough to warrant cessation
therapy. Dyspepsia and abdominal pain have also occurred. As with s
penicillins and some other cephalosporins, transient hepatitis and cho
tatic jaundice have been reported rarely.

Hypersensitivity—Allergic reactions in the form of rash, urticaria,
edema, and, rarely, erythema multiforme, Stevens-Johnson Syndrom
toxic epidermal necrolysis have been observed. These reactions usu
sided upon discontinuation of the drug. Anaphylaxis has also been re

Other reactions have included genital and anal pruritus, genital mo
vaginitis and vaginal discharge, dizziness, fatigue, and headache Rev
interstitial nephritis has been reported rarely Eosinophilia, neutropeni
thrombocytopenia, and slight elevations in SGOT and SGPT have bee
reported

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ZOVIRAX®
(acyclovir) CAPSULES

Prevent genital herpes recurrences month after month with daily therapy.

(In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.)

Please see last page of this advertisement for brief summary of prescribing information.

ZOVIRAX®
(acyclovir)
CAPSULES

Help free your patients from recurrences.

Daily therapy

Coping with genital herpes is rarely easy. For some, the worst part is the pain and discomfort of frequent attacks — month after month, year after year. For others, the emotional burden presents a more difficult problem, leading to social isolation, anxiety, and diminished self-esteem.

Prevent or reduce recurrences

Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

Generally well tolerated

Daily therapy with ZOVIRA CAPSULES is generally wel tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/ vomiting, vertigo, and arthralgia.

The physical and emotiona difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episode as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRA CAPSULES.

Please see brief summary of prescribing information on next pa

Prevent recurrences month after month*

ZOVIRAX®

(acyclovir) CAPSULES

Brief Summary

NDICATIONS AND USAGE: Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of utaneous or systemic involvement. These factors hould determine patient management, which may nclude symptomatic support and counseling only, r the institution of specific therapy. The physical, motional and psycho-social difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician hould determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in reating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

First Episodes (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

Recurrent Episodes:

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

CONTRAINDICATIONS: Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

WARNINGS: Zovirax Capsules are intended for oral ingestion only.

PRECAUTIONS: General: Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

Drug Interactions: Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the F_1 generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

Pregnancy: ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS — Short-Term Administration: The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

Long-Term Administration: The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

Chronic suppressive therapy for recurrent disease: One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

Intermittent Therapy: One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

Patients With Acute or Chronic Renal Impairment: One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance ≤ 10 ml/min/1.73/m².

HOW SUPPLIED: Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200" - Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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*In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

Burroughs Wellcome Co., Research Triangle Park, North Carolina 27709

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Program Notes
Arctic Investigations Laboratory
Centers for Disease Control

Partial Penicillin Resistance (PPR) Pneumococcal Isolates Recovered from Alaskan Patients

National surveillance of pneumococcal serotypes and antimicrobial sensitivities by the Centers for Disease Control indicates that the overall incidence of decreased or partial susceptibility of penicillin (PPR) in pneumococci is still quite low (3.7%) despite reports of isolation rates of 15% from a few U.S. areas and up to 65% from some South African Hospitals. Pneumococci completely resistant to multiple antibiotics including penicillin have been isolated from up to 11% of blood isolates and 30% of carriage isolates of inpatients from Europe and South Africa but have been only sporadically reported in the United States. The suspicion that partial penicillin resistance heralds higher grade antimicrobial resistance in a given geographic area appears to be fact.

A pilot laboratory-based surveillance project of all invasive pneumococcal disease was established here in Alaska by the Arctic Investigations Laboratory (AIL) in 1980. Pneumococcal isolates were obtained from cultures of blood and CSF from both Native and non-Native patients from Anchorage and the Yukon-Kuskokwim Delta Region in southwestern Alaska. All pneumococcal cultures sent to AIL were confirmed as *S. pneumoniae* by Optochin sensitivity and were screened for sensitivity to penicillin by disc diffusion using a 1 ug Oxacillin disc. All cultures were forwarded to the Division of Bacterial Diseases, CDC, Atlanta, for serotyping and antimicrobial MIC determinations. This surveillance system was extended to all hospitals in Alaska in January 1986 when data suggested marked regional differences between those two areas.

During 1980-84, 37 isolates were received from the Yukon-Kuskokwim Delta Regional Hospital for serotyping and antimicrobial sensitivity testing. Of these, 10 (27%) were found to be partially resistant to penicillin with MICs between 0.1 and 1.0 ug/ml. Only one isolate from this region was from a non-Native patient. All PPR isolates were serotypes currently included in the pneumococcal vaccine. A majority were typed as 19A, a serotype frequently identified elsewhere as a pathogen showing full penicillin resistance. In contrast, less than 4% of 29 isolates from Alaska Native Medical Center and only one of 44 isolates from other Anchorage area hospitals were partially resistant. Most of the PPR isolates (90%) were recovered from infants less than two years of age.

The primary clinical presentations of patients with PPR disease included pneumonia (7 cases), meningitis (1), otitis media (1), and sepsis with no other focus of infection (1). Associated illnesses present in eight patients included congenital defects, recurrent pneumonia, cancer of the colon, anemia, immunodeficiency, and tuberculosis. Two deaths occurred among patients with PPR isolates: a two-year old with an immunoglobulin deficiency and an adult with colon cancer. Both isolates were vaccine serotypes and had MICs by agar dilution of 0.25 ug/ml.

During 1986, a total of 86 invasive isolates were sent to AIL from all but six hospitals statewide bringing the seven year total of PPR isolates confirmed with MICs to 15 of 284 total invasive isolates screened by Oxacillin discs. In the absence of suitable automated standardized methods for determining antibiotic MIC levels for pneumococci in clinical laboratories, it is recommended that all isolates recovered from normally sterile body sites be screened with a 1 ug Oxacillin disc.

The calculated accuracy of oxacillin disc screening determined in parallel with MICs for 131 Alaskan isolates is summarized in Table 1:

Table 1.
Accuracy of Oxacillin Disc Testing compared to Penicillin MIC of ≥ 0.12

Zone Diameter:	$\leq$ 19mm	$\leq$ 15mm
Sensitivity	87%	87%
+Predictive value	42%	77%
False + rate	58%	23%
Specificity	85%	97%
-Predictive value	98%	98%
False - rate	2%	2%

In order to assess the current prevalence of resistant pneumococci, a survey of pneumococcal isolates recovered from nonsterile sites (sputum, throat swabs, nasopharyngeal swabs) was recently undertaken at the Alaska Native Medical Center. Of 155 isolates screened for penicillin sensitivity by Oxacillin disc diffusion, 26% had partial or complete penicillin resistance with inhibition zones of ≤15mm. This indicates that pneumococci with reduced penicillin sensitivity, and possibly other antibiotics as well, are common in Anchorage. Surveys of other regions and populations are in progress.

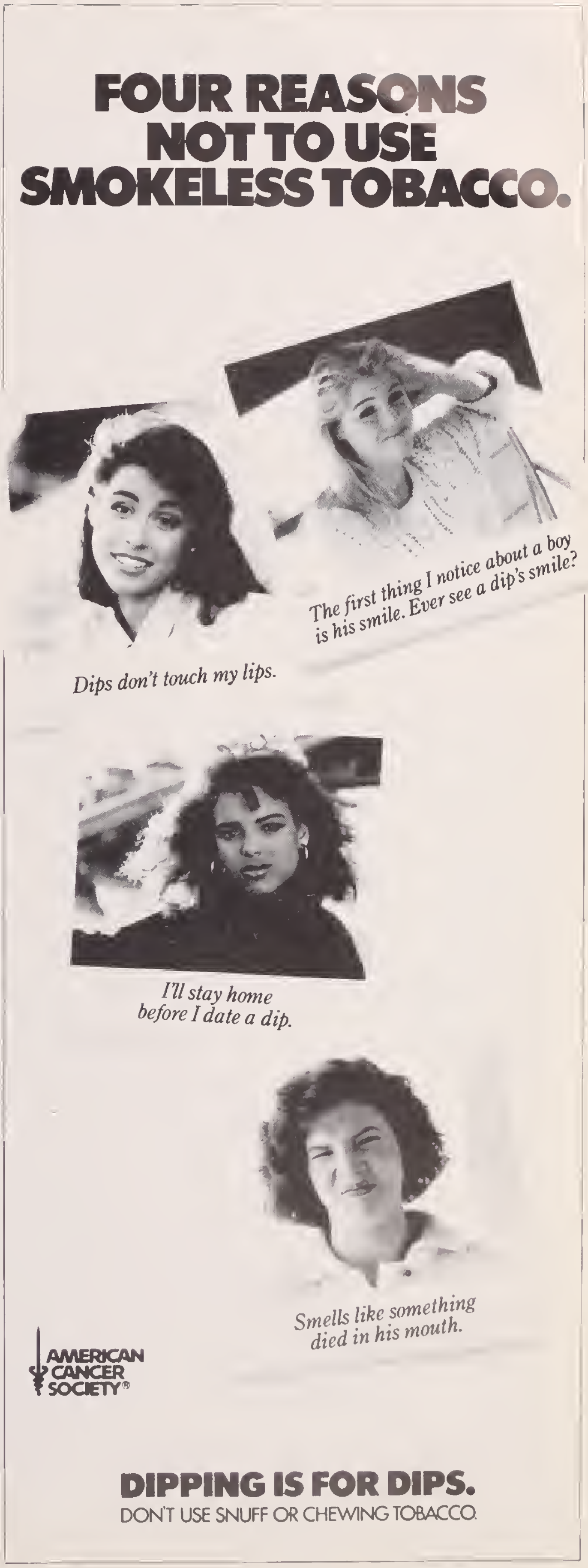
Clinicians should be aware that screening invasive pneumococcal isolates is relatively simple and indicated. All pneumococcal isolates from normally sterile body sites should be referred to AIL for confirmation and MIC testing. This is currently performed by both agar and broth dilution techniques and results on organisms screened as partially or completely resistant will be quickly available to assist with patient management. Penicillin resistant infections should be considered when patients do not improve as expected on B-lactam antibiotics. In addition, prior hospitalization, recurrent pneumonia within the past year, and recent B-lactam antibiotic use appear to be factors predisposing to disease with pneumococci fully resistant to penicillin (1).

Isolates, particularly from CSF screened for penicillin resistance using the Oxacillin disc method with inhibition zones of ≤15mm should be considered to have decreased sensitivity to penicillin and alternative or additional antimicrobial agents such as chloramphenicol should be considered. High dose penicillin G may still be used to successfully treat penicillin-resistant pneumococci with MICs <2.0 ug/ml At this time, penicillin should continue to be the drug of choice for patients with community-acquired pneumococcal pneumonia who are not critically ill (1). Blood cultures are strongly urged for diagnostic confirmation in all patients with pneumonia and pneumococcal vaccine should be given to all high risk individuals.

Penicillin resistance of pneumococci appears to be related to structural alternations in cell wall penicillin-binding proteins. AIL is currently studying mechanisms and risk factors for, and the epidemiology of, partial penicillin resistance in Alaskan patients.

REFERENCE

1. N Engl J Med 1987;3117:18-22.


FOUR REASONS NOT TO USE SMOKELESS TOBACCO.

The first thing I notice about a boy is his smile. Ever see a dip's smile?

Dips don't touch my lips.

I'll stay home before I date a dip.

Smells like something died in his mouth.

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My Turn

Pediatrics, Providence, and Progress

How did the Towers get to Anchorage in the first place back in July of 1954? I had a favorite grandfather, an EENT surgeon in Vermont, who had visited Alaska in 1928 and had done a great deal of hunting and fishing as a guest of President Coolidge's Secretary of the Interior. I'd seen the black and white photos of his expeditions while I was growing up, and I suppose I was subconsciously bitten with the desire to go. When Betsy and I were finishing up our pediatric and internal medical residencies at Yale we wrote, asking for information about the area, to one of the most remarkable Alaskans we've ever known, C. Earl Albrecht, M.D., then Commissioner of Health for the Territory of Alaska. He wrote back immediately, offering strong inducements including consulting positions with the Department of Health and the new Bureau of Indian Affairs Hospital (now the U.S.P.H.S. Alaska Native Medical Center). He followed this up with a personal visit with us in New Haven and the die was cast; we were off for Alaska via the highway two months later.

We arrived, dusty but excited on a beautifully pristine July day, simultaneously with a most remarkable pediatrician and shortly to be partner, the late Helen S. Whaley, M.D. who had been lured by the same bait thrown out by Dr. Albrecht. Office space was nonexistent so we plunked down our savings for an old (and now historic) former railroad house on the corner of Second Avenue and not only lived there but started seeing patients there almost immediately. There had been no pediatricians in private practice in Alaska up to that point, and Helen (then working for the Alaska Medical and Surgical Clinic) and I created something of a stir since old Providence Hospital down on "L" Street was rapidly filling up with patients from a severe polio epidemic.

Orthopedist William J. Mills, M.D., arrived shortly thereafter, and we were all swamped trying to contend with over 20 odd patients in iron lungs. (My oldest daughter Chris, then just a yearling, would gleefully play with the kids on the waiting room floor until I could get to them, tap, and admit them with bulbar polio.)

We wasted no time in getting appointed to the Providence Hospital staff and, as I recall, I was the first Chief of Pediatrics, a position Helen and I rotated yearly until Dr. Harvey Zartman joined us three years later. There were perhaps ten available beds on the north end of the first floor if we shoehorned cribs and bassinets into every available space. In those pre-Salk and pre-Sabin vaccination days and pre-measles, German measles and mumps immunization times, we were loaded with sick kids; meningitis, encephalitis, osteomyelitis, and severe croup and bronchiolitis epidemics. There was a nursery upstairs but no isolation nursery and nothing even resembling a premature baby facility. There were a couple of ancient tub-like heaters for premies and pretty soon some Armstrong incubators.

The Sisters of Providence of that day ran a tight ship moneywise and found it difficult to make many changes or improvements: the money was not at hand. As physician specialists in ENT, surgery, orthopedics, internal medicine began to come to the expanding territory, they often had to contribute the necessary funds to buy the tools they needed. There were no radiologists or pathologists, so we read our own films and did our own autopsies.

Helen and I worked out of our, now crowded and not very well heated, old railroad house on Second Avenue. We had very long hours each day and no E.R. doctors to see patients at night. We even made house calls, at least I did until I wandered into the wrong house late one night looking for a croupy child and found a scantily clad woman looking for a different nocturnal visitor!

We hadn't been here but a week or so before investigating the pediatric needs at the Bureau of Indian Affairs new tuberculosis hospital. There were only two doctors there and over 75 terribly sick children with illnesses that I'd only read about in the text books. Helen and I, after bulldozing through some governmental red-tape, took to making evening calls there before midnight. We would see the kids brought in by bush plane at sunset and not infrequently dead by morning from typhoid, dehydration, meningitis, or miliary tuberculosis. It was very exciting and very rewarding. We became deeply enamored of Alaska and of Alaskans, frequently people from the upper Midwest, Minnesota, North Dakota, Washington and Oregon. Statehood fever was in the air, and we loved it.

With statehood came winds of change. The Anchorage medical community of the early fifties numbered just 28 doctors including the Indian Hospital and the military hospital (just 28 lawyers too!). The Sisters of Providence little-by-little began to harken to the pleas of a very capable staff of doctors, well-trained and independent and anxious to provide for their in-house patients all the latest.

The Presbyterian Hospital appeared on the scene in direct competition and shortly after, the Sisters made the remarkable jump to their current site out on Providence Drive. The hospital seemed too far away from the center of things, but it was magnificent and compelling when it absorbed the shock of the terrible '64 earthquake. Providence Hospital and its medical, nursing and administrative staff have had few finer hours. The hospital's role in this disaster was magnificent, beyond the wildest dreams or hopes of conscientious experts practicing disaster drills.

Prosperity, and the resulting boom and growth, picked up after the earthquake. New physicians and many more deserving patients made even the new Providence facilities inadequate for the need. We now had a dozen fine pediatricians, and we pushed for a better nursery, an up-to-date premature baby nursery and our own pediatric floors. Thanks in large part to Sister Barbara Ellen's leadership, Dr. Dion Robert and I were able to get a newly designed premature baby ward opened, the first beginnings of Dr. Jack Jacobs' Newborn Intensive Care Nursery.

Now with the Project 90's embellishments complete, we pediatricians luxuriate not only in a first-rate nursery, up-to-date obstetrics and pediatrics facilities, but we even enjoy a fine pediatric intensive care ward. The pediatric nurses now do what we doctors used to spend long midnight hours trying to accomplish; getting blood specimens from tiny or very sick children and getting I.V. fluids started and running. Providence has steadily improved its image as the referral center for Alaska.

I can't claim to have been very visionary about these necessary changes in the big picture of medicine in Alaska. I never foresaw I would be in the midst of a thriving energetic western boom town of 200,000 plus. Like many of the immigrants to Alaska in pre-statehood days, I got a tremendous kick out of the life and times then and am a bit more nostalgic about the memories. It's too easy perhaps to forget the long nights of work and anxiety, and the lack of proper facilities, equipment and personnel.

The physicians of the early Providence Hospital days worked in tandem with the Sisters of that day and as time passed we all came to appreciate that they were a class act. We began to understand that, in the end, they had the foresight and leadership, amplified by current administrator Al Camosso, to provide Alaska with a superb hospital to deliver the health care it needed.

John Tower, M.D.

Dr. John Tower with two of his young patients.

President's Page

I would like to bring you an update on the happenings of the ASMA Executive Committee meeting of October 23. The main objective of the meeting was to give the first annual efficiency report to Ray Schalow, our executive director. It was the unanimous feeling of the members of the executive committee that Ray has done an outstanding job since coming aboard in September of 1986. The summary of the report was as follows:

> "The executive committee members have been impressed and gratified with the speed which you have gone from ground zero in medical knowledge to your present position as a very able spokesman for ASMA. Your enthusiasm is appreciated. We feel that one of your best strengths has been the management ability shown in turning the office morale around plus getting the financial picture well-clarified. We feel that you have done an outstanding job for your first year. We look forward to working with you in the ensuing years. In other words, we want you to stay around."

The Fall Council meeting of ASMA was held on October 24. Considerable ground was covered in a short period of time. One of the noticeable committee reports was that of the Accreditation Committee. Marion Witt has done a superb job in the past two years in getting the Accreditation Committee fully accredited again. She has developed a policy manual for the Accreditation Committee that was approved by the Council. Our thanks and appreciation go out to Dr. Witt for her tremendous effort.

Dr. Charles Aarons, formerly of Dillingham, gave a very interesting talk on the perils of private practice in rural Alaska. As most of you may know, he has been forced out of private practice in Dillingham by the Bristol Bay Area Health Corporation. They refused him hospital privileges because he did not have malpractice or liability insurance. Some of the questions that he brought before the council have been referred to the Bush Medicine Committee for further research.

Dr. Charles Steiner is chairman of the CME committee for the annual meeting to be held in June 1988 in Fairbanks. He has certainly taken charge of the committee and has put a lot of energy into developing a very excellent CME program.

Dr. David McGuire, chairman of the TORT Reform Committee; Rick Urion, lobbyist; and Ray Schalow, Executive Director, gave us an update on the initiative process. As you know, part of the initiative has been declared unconstitutional by the Attorney General's office. The Citizens' Coalition for Tort Reform is going to proceed with an alternate initiative regarding just the evolution of the doctrine of joint and several liability. Dr. McGuire feels that the substitute initiative will be approved by the legislative body of the state. Again, sponsors are needed, and signatures are going to be needed. We need your efforts.

Representative Kay Brown of Anchorage was a guest speaker at the council, discussing her Parental Leave Bill, HB-224. There were some very intriguing questions asked of Representative Brown, such as what penalties will be placed on a business that does not comply with the bill, etc.. There will be a follow-up report on this bill at the next council meeting.

Dr. Don Thieman gave a report on the Young Physicians Committee. He plans to set up a meeting of young ASMA physicians at the annual meeting in Fairbanks.

There was a very heated discussion regarding the medical directory for 1989. We debated if the directory should be for ASMA members only or if all the physicians in the state be listed in the directory, whether they are members of ASMA or not. The motion was made that we continue publishing the directory as is, which will include non-members of ASMA. This motion was passed.

Other areas covered discussions on the proposed veterans hospital, and a proposal was approved by the Council to create an institutional membership to include a Councilor-At-Large position for the public health service.

The next council meeting will be held in Juneau, February 10-12, 1988. We look forward to having an exciting meeting there. Hopefully, we will be able to do some successful lobbying.

As you can see, a great deal was covered during this council meeting. Needless to say, I have not mentioned all of the committee reports nor all of the other items that were covered. If you, as a physician in the state of Alaska, have any items of interest to be brought to the public, we welcome your input at our next council meeting.

Jerry Little, M.D.
President
Alaska State Medical Association

Auxiliary News

Alaska State Medical Association

In 1645, one vote gave Oliver Cromwell control of England.

In 1649, one vote caused Charles I of England to be executed.

In 1776, one vote gave America the English language instead of German.

In 1845, one vote brought Texas into the Union.

In 1868, one vote saved President Andrew Johnson from impeachment.

In 1875, one vote changed France from a monarch to a republic.

In 1876, one vote gave Rutherford B. Hayes the presidency of the United States.

In 1923, one vote gave Adolph Hitler leadership of the Nazi party.

In 1941, one vote saved Selective Service just weeks before Pearl Harbor was attacked.

In 1973, one vote, cast by the Vice President of the Senate, broke a tie that would have prevented construction of the Alaska pipeline.

tion of the Alaska pipeline.

In 1986, one vote in the Alaska State House killed meaningful tort reform.

Two years ago a legislator was vacillating on his support of tort reform. In fact, he was leaning against it. One phone call to him and one hour spent talking to him convinced him of the seriousness of the liability crisis. He is now a strong supporter of tort reform.

The point is that YOU can make a difference. One letter written to your representative, one phone call, one hour spent explaining tort reform to a member of the public will bring about our goal.

The legislature will soon be in session, and I urge each and every one of you to get involved. YOU will make a difference.

Cassie Hill
President
Alaska State Medical Association Auxiliary

A defense against cancer can be cooked up in your kitchen.

There is evidence that diet and cancer are related. Follow these modifications in your daily diet to reduce chances of getting cancer:

1. Eat more high-fiber foods such as fruits and vegetables and whole-grain cereals.

2. Include dark green and deep yellow fruits and vegetables rich in vitamins A and C.

3. Include cabbage, broccoli, brussels sprouts, kohlrabi and cauliflower.

4. Be moderate in consumption of salt-cured, smoked, and nitrite-cured foods.

5. Cut down on total fat intake from animal sources and fats and oils.

6. Avoid obesity.

7. Be moderate in consumption of alcoholic beverages.

No one faces cancer alone.

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Editorial

Recently, I stumbled upon an old report on health care in Alaska. Written in 1954, the Parren Report caught my eye first because of its crumbling, yellow-stained papers that seemed determined to burst their weakened binding and go flying across my office, and second, because on one of the pages was a population histogram made from the 1950 census—the first I had seen in my 12 years in Alaska. Now, I am an epidemiologist, which has been described as "a person in search of a denominator," and have spent a vast amount of energy trying to find accurate population numbers for my own work. Suddenly my half-hearted interest in the report turned to concentrated effort.

The Parren Report was one of the most influential reports about health care in Alaska's history. We can trace the roots of our current health care delivery system to problems it identified and to the solutions it recommended—many of which were implemented. The report documented a disgraceful state of affairs in the health of Alaska's Native peoples and a failure of the U.S. Government to discharge its obligations to care for its citizens in Alaska. The report summed up that deplorable situation by calling Alaska "Unfinished Business."

As a result of the report's findings, sweeping changes were made in Alaska's health care system. One of the most profound was the transfer of responsibility for health care from the Department of the Interior to the Department of Health, Education and Welfare and the U.S. Public Health Service, Bureau of Indian Affairs. An all-out attack was begun to fight tuberculosis, responsible for 24% of all Alaskan deaths in 1950. Major efforts were begun to improve mental health services and to establish village-based health services.

The Parren Report is worth careful study as we begin to face the many challenges of providing health care services to all Alaskans in the future.

- We must deal with liability, insurance, compensation, and negligence;
- We must prepare for the existence of four major $100 million-plus health care facilities in Anchorage—because a new Alaska Native Medical Center and a new Elmendorf AFB-VA hospital appear to be certainties;
- We must develop policies to deal with all issues surrounding AIDS, including confidentiality, screening, contact referral and assistance, education and behavior modification, accessibility of health care services, financing, and ethical obligations of all health care workers to care for those infected with the AIDS virus.

Our memories are short. We must learn from the past. Although many of the problems we face are different from those Alaskans faced in 1950, still, the principles and policies that were guiding health care in 1954 have great relevance today. Our many agencies, institutions, organizations, and governmental programs must identify shared principles and goals—and they must work together to identify solutions. It is time to finish Alaska's "Unfinished Business."

New Feature Presents Early Alaskan Medical Texts

Beginning with the current issue, *Alaska Medicine* will include a new feature entitled "Glimpses of Alaskan Medical History." Each one-page article will present an historical vignette in the form of an original text, briefly introduced and annotated by Robert Fortuine, M.D.. Over the next few years this column will present early Alaskan writings on medicine, health, and disease written by physicians, explorers, naturalists, traders, missionaries, and government officials.

We welcome this new feature to the journal and hope that readers will not only find it interesting, but also a means of enriching their knowledge of Alaska and its medical heritage. We are indebted to Dr. Fortuine for contributing this fascinating series to *Alaska Medicine*.

Letters to the Editor

Dear Editor:

That once far off tomorrow is rapidly approaching for us and the time has come to consider things more realistically, putting aside our unfinished hopes and dreams. The time as arrived to sort, save, sell, donate and destroy. One way of putting it is "Blessed be Nothing" and another is "That There Is Just So Much That One Can Take To That Cell-like Room in the Pioneer Home."

Not finding a buyer, we began some months ago by giving away between $30,000.00 and $40,000.00 worth of ophthalmological and otolaryngological surgical instruments and equipment. These were given to the Central Peninsula General Hospital in Soldotna, Alaska where my husband had been made so welcome when we moved from Anchorage to our homestead on the Kenai Peninsula. However, we are still wondering where to place the contents of a fully equipped ophthalmological diagnostic and treatment room and of a partially equipped otolaryngological room.

The question really is where to put all of the rest. The Loussac Library in Anchorage has asked for my husband's private and public papers which he had intended to offer to the University of Alaska.

At present I am sorting through seven, 4-foot shelves of Milo's publications and reprints; 42 shelves four-foot long of mostly business correspondence—to and from—dating back to the 1920s or perhaps earlier; 8 file drawers pertaining to Milo's political life; 149 shelves 2'5"-long of the individual medical records of the. 57,526 patients seen between 1948 and 1984. Many talks and papers have been prepared from them but there is much more material for socio-medical writing possible from them. Last, but not least, there are 64 years of carefully kept diaries.

In addition there is a four-drawer file of the activities of the Eye, Ear, Nose and Throat Foundation and it's Eye Bank which we began in the early 1950s. One four-drawer file containing a folder for each year of the 100 years that he lived of the artist who did most of the paintings in our collection, as well as photographs, and material pertaining to his life and art. A considerable amount of correspondence with his relatives in Germany (mostly in German) dating back to the late 1800s. Most precious of all is the Thank-you signed by/for 100 children of Yugoslavia to whom he sent Care packages during the War when he was not allowed to send to his own family in Germany.

Much of the above will be useful in writing up the life of Milo H. Fritz, M.D.—Doctor to Alaska. Some of it is and will be interesting reading. Nobody needs 30 years of laundry receipts or invoices for nearly every purchase for 50 years. I am sorting through it all, an interesting endeavor that brings back many memories. What I am saving and putting into chronological order making it easier for any use in the future.

In what was the patient reception area I have eight, 8-foot tables set up with cardboard boxes on them labeled by year into which I toss loose papers, photographs, souvenirs, clippings, publications, etc. preparatory to filing them properly.

Besides all of the above there are more than sixty scrapbooks of memorabilia of the life and times of Betsy and Milo Fritz.

As I carry on this work, and not just because these are cherished by us, it occurs to me that what is needed is a museum or a section of an existing museum dedicated to the history of Medicine, Nursing and Public Health in Alaska.

What we have could be the genesis of such.

Besides what I have already mentioned, we have an interesting display of the Eye, Ear, Nose and Throat Foundation and the Eye Bank even to the Eastern Airlines Thermos in which was shipped the first donor eye for the first and successful corneal transplant in our state. We have a small collection of antique instruments, chiefly ophthalmological. There are maps of Milo's trips into the bush to care for those in need who could not come into town. We have a lovely display of gifts from grateful patients some of which are very valuable works of art or Alaskan artifacts. A considerable number of slides and photographs of one man's 47-year medical career in Alaska as a private physician from Ketchikan to Barrow and from the Canadian border to the Pribilof Island; as an Army officer, a Command Surgeon he covered the Territory to care for the soldiers and airmen; at times he was a contract physician to the health agencies; as a pilot; as a writer; as a three term Representative in the State Legislature; as an effective office holder in the local and state medical association; as an educator.

There are diplomas, certificates, awards, medals, decorations, and recognitions of his service to his fellow man. There is his not inconsiderable medical library and my small nursing library.

As the first president of the Alaska Nurses Association, I have some material from those years of infancy, the fight for licensure for nurses, the stormy time of labor relations and a more realistic pay scale for nurses.

And, of course there is the 8' x 4' mural by Fred Machetanz and the accompanying preliminary sketch and paintings as well as the artist's framed explanation of the work as was on display at the Anchorage Fine Arts Museum before it came to us. This tremendous and valuable work, entitled by the artist "A Medical Saga," depicts Milo's life in Alaska.

Last but not least among our 1,500 or so paintings, there are a few pertaining to Milo and medicine, i.e., a prize winning oil of the Berkshires by Milo's father, Henry E. Fritz, Ph.D., which he painted while his wife was going through a long and difficult labor when Milo was born. One is of the garden where Milo played as a youngster. Another of Milo as a lifeguard earning his way through medical school. Yet another, of the Duke University Chapel frequented by Milo while an eye, ear, nose and throat intern and resident at Duke Hospital—one which always hung appropriately in the office waiting room of Christ Healing the Blind. This copy was done by Milo's father as a gift when Milo began private practice. The original by Ledovico Carracci, 1555–1619, hangs in the Ringling Museum in Sarasota, Florida. We have a Vernon A. Cates, M.D., painting and though it does not have a medical theme, it has a very definite medical connection. Lastly there is a Henne, her first large canvas, which she called "The Crisis" and graciously changed to "The Consultation" to please us. Using artifacts from the museum in Juneau, she showed three shaman consulting over an obviously very ill Native man.

All of the above is a great deal of I, my, and me but there must be a prodigious amount of the same sort of material all over the State which deserves to be in a museum before it is lost.

As an example, I recall how in 1961 en route home from weeks on a bush clinic, I was in Fort Yukon between planes. As I waited for the air taxi to take me to Fairbanks, I walked about this historic place. It was the day before Mrs. Grafton Burke, widow of the doctor who came to Alaska in 1906 and in 1916 had founded the Archdeacon Hudson Stuck Memorial Hospital in Fort Yukon where he cared for the sick until 1938. Mrs. Burke and many others had worked so hard to raise funds to equip the hospital. She was returning after many years absence to join in the Centennial Celebration of the Anglican Communion in the far north. As I walked about that summer day I saw the contents of that now closed hospital scattered all over, thrown haphazardly into the tall grass. Great preparations were being made for the celebration, but how heartbroken she would be to see to what end her efforts had come. Who can say what treasures were lost and what interesting material would have been available in the medical records.

I digress only to show one example of what could have been an interesting exhibit.

In the Alaska Magazine and in the Alaska Churchman there have been many articles about doctors and nurses, documenting not only their valuable service but interesting as well. I am sure that other faiths have many such examples—the Moravians in the Bethel area where and with whom Dr. Romig served, the Catholic Sisters who went to the Nome area so long ago, and the Presbyterians in the Southeastern part of Alaska.

I was going to name some of those living who have contributed so much but fear I might overlook some. But I am sure that the reader has many in mind who might be mentioned for studious or heroic deeds who must be remembered in a museum such as I feel is needed.

There is much to tell and much to save. If the material is not asked for, it is scattered, lost, destroyed or even sent out of the state.

Are there not some among us who would be interested in such a project dedicated to the History of Medicine, Nursing and Public Health in Alaska?

(Mrs.) Elizabeth Berry Fritz
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Otitis media due to *S pneumoniae, Haemophilus influenzae*, staphylococci, streptococci, and *Neisseria catarrhalis*

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Contraindication: Keflet is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: BEFORE CEPHALEXIN THERAPY IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS AND PENICILLIN. CEPHALOSPORIN C DERIVATIVES SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS

SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES

There is some clinical and laboratory evidence of partial cross-allergenicity of the penicillins and the cephalosporins. Patients have been reported to have had severe reactions (including anaphylaxis) to both drugs.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics cautiously. No exception should be made with regard to Keflet

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should be ruled out.

Usage in Pregnancy—Safety of this product for use during pregnancy has not been established.

Precautions: *General*— Patients should be followed carefully so that any side effects or unusual manifestations of drug idiosyncrasy may be detected. If an allergic reaction to Keflet occurs, the drug should be discontinued and the patient treated with the usual agents (eg, epinephrine or other pressor amines, antihistamines, or corticosteroids)

Prolonged use of Keflet may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug

Keflet should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended

Indicated surgical procedures should be performed in conjunction with antibiotic therapy

As a result of administration of Keflet, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis

Usage in Pregnancy—Pregnancy Category B— The daily oral administration of cephalexin to rats in doses of 250 or 500 mg/kg prior to and during pregnancy, or to rats and mice during the period of organogenesis only, had no adverse effect on fertility, fetal viability, fetal weight, or litter size. Note that the safety of cephalexin during pregnancy in humans has not been established

Cephalexin showed no enhanced toxicity in weanling and newborn rats as compared with adult animals. Nevertheless, because the studies in humans cannot rule out the possibility of harm, Keflet should be used during pregnancy only if clearly needed

Nursing Mothers— The excretion of cephalexin in the milk increased up to 4 hours after a 500-mg dose, the drug reached a maximum level of 4 μg/mL, then decreased gradually, and had disappeared 8 hours after administration. Caution should be exercised when Keflet is administered to a nursing woman

Adverse Reactions: *Gastrointestinal*— Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely. The most frequent side effect has been diarrhea. It was very rarely severe enough to warrant cessation of therapy. Dyspepsia and abdominal pain have also occurred. As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely

Hypersensitivity— Allergic reactions in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens-Johnson Syndrome, or toxic epidermal necrolysis have been observed. These reactions usually subsided upon discontinuation of the drug. Anaphylaxis has also been reported

Other reactions have included genital and anal pruritus, genital moniliasis, vaginitis and vaginal discharge, dizziness, fatigue, and headache. Reversible interstitial nephritis has been reported rarely. Eosinophilia, neutropenia, thrombocytopenia, and slight elevations in SGOT and SGPT have been reported

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Prevention of hypokalemia requires particular attention in patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis and ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy, certain diarrheal states, or other states where hypokalemia is thought to represent particular added risk to the patients
In patients with hepatic cirrhosis and ascites, sudden alterations of electrolyte balance may precipitate hepatic encephalopathy and coma Treatment in such patients is best initiated in the hospital with small doses and careful monitoring of the patient's clinical status and electrolyte balance Supplemental potassium and/or spironolactone may prevent hypokalemia and metabolic alkalosis in these patients
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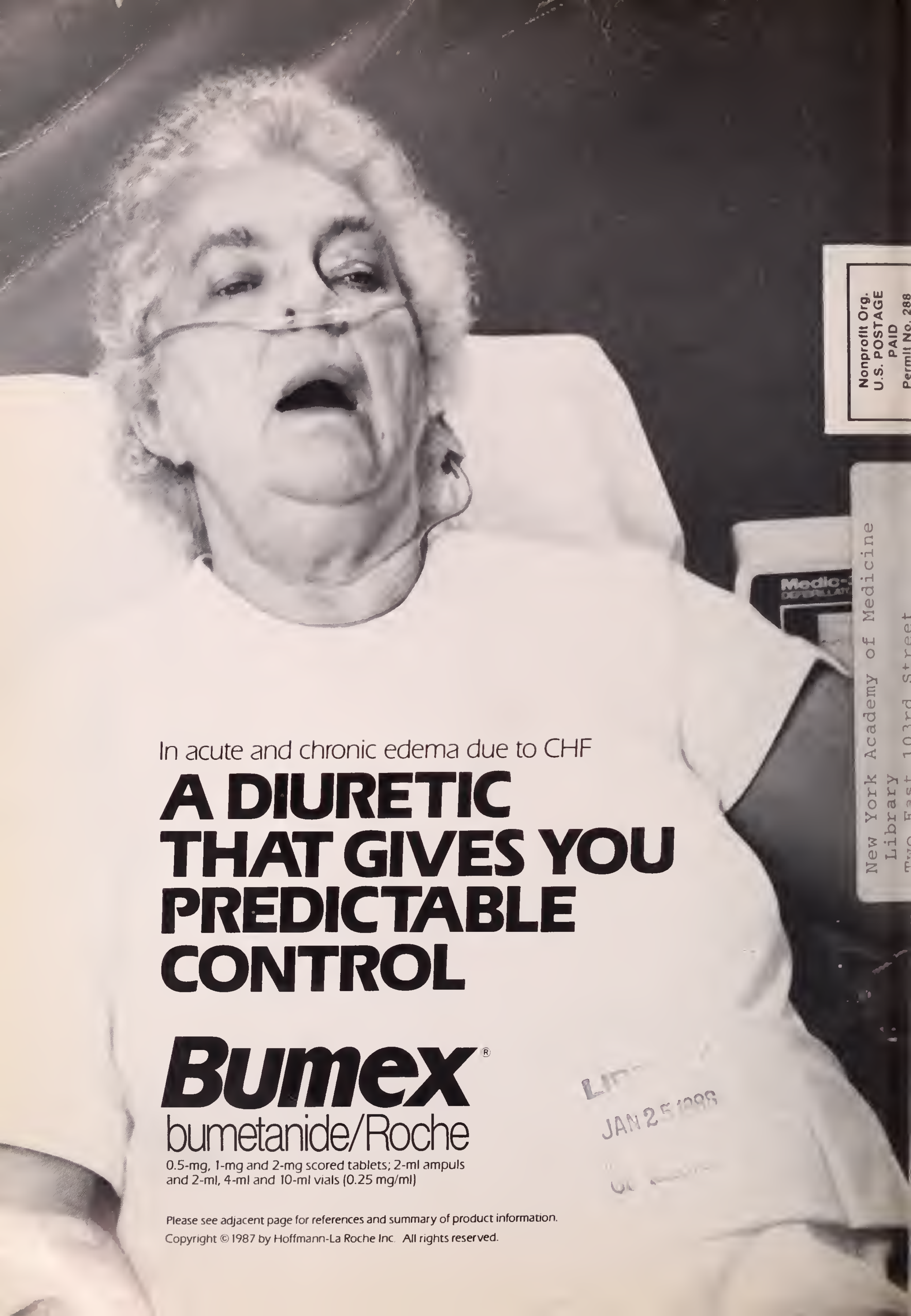

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Reflections in Ice. Graciously furnished by John Hyde, Alaska Department of Fish and Game, Juneau, Alaska.

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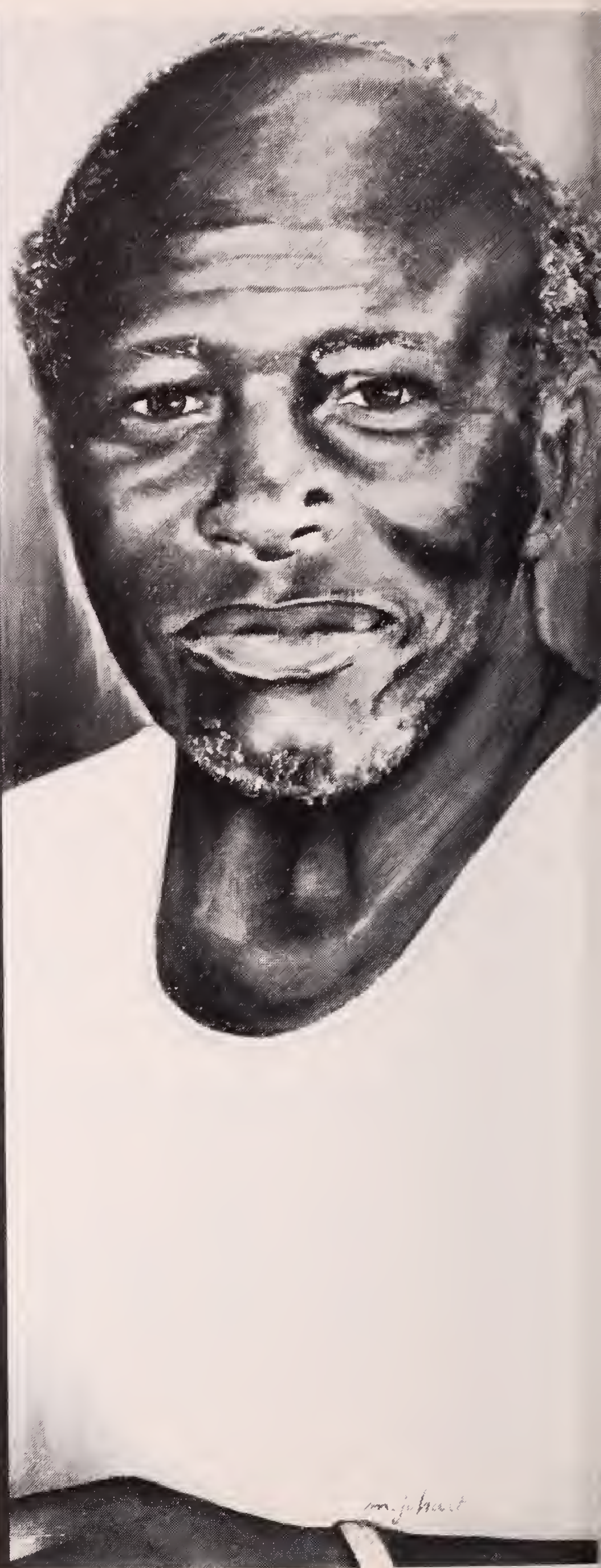

Why is There a Need to Have a Rational Process to Develop Public Health Policy?

by Robert (Bert) Hall[1]

In my opinion, there could have been no better theme chosen for this year's Congress than what has been selected, TOWARD A STATE PUBLIC HEALTH POLICY FOR THE NINETIES—ALASKA'S UNFINISHED BUSINESS. For those of us who claim to work in the field of Public Health or for the health of the public in Alaska, there can be no more important an agenda for the next few years than to get involved with and influence the development of a process leading toward that goal.

If recent years are any predictor of the future, it is not only important but critical that we enter into such a venture, and it is absolutely imperative that we are successful. Whether we realize it or not, we are already losing a battle by default that some may not even know we are in. There is virtually no one else to take up the gauntlet of Public Health—there is no other organized force that cares enough to see that we even hold our own.

Let me quote a few sentences from Saturday's Anchorage News headline story on the 1988 state budget: Prudhoe Bay oil producers implored the state to preserve a $93 million severance tax cut. Yes, the Senate said. Senior citizens urged lawmakers to leave intact the $47 million Longevity Bonus Program. O.K. said the House. Lobbyists for electrical utilities, labor unions and contractors stroked their favorite beast, the $350 million Bradley Lake Hydroelectric Project, and lawmakers are preparing to give the go-ahead to the biggest construction project in state history. School districts asked lawmakers not to take too big a bite out of state money to pay off school construction debt. The House, at least, is ready to oblige them. Cities lobbied hard against big cuts in revenue sharing and municipal assistance. And the cuts won't be too awful big, the cities are being told. Public employees fought a $40 million reduction in wage and benefit costs, and the Senate, at least, appears ready to pay the money.

Hear anything about Public Health? Even the most casual observer has to note that in these days of declining revenue, it's either the squeaky or political wheel that gets the grease, or there is ongoing support for those programs where clear state policy has predetermined that certain services and programs will be protected—at least to a degree.

The only purpose in my presentation is to reaffirm what you already know—there does not now exist any clear state Public Health Policy. If we do not take the leadership role, there will certainly be none for the 90s. My secondary purpose is to get you excited - or angry - or defiant - or vocal - or just ticked off enough that you will make a personal commitment to get involved.

You know, we have taken a shot at this before — in fact, there have been several missed opportunities. Six years ago, ALPHA sponsored a similar Congress with almost an identical theme. We called it "Toward a State Health Policy for the Eighties." It was sort of a tongue-in-cheek suggestion to state decision-makers that we were already into the 80s, but there was no state policy in place any more than there was even a sense of direction—and all at a time when it appeared that oil dollars would be flowing into the treasury for years to come. In its own way, it was a good conference. It provided a forum to get a general look at several specific policies and to review in general a number of specific programs with many of the state's key administrative leaders in the same room at the same time.

Where it missed the target, however, was in the title's suggestion of developing a state health policy. In fact, legislative actions in following years seem to have wandered even further from what had been assumed policies for distribution of state resources. Perhaps we hoped for too much, too fast. Perhaps we should have pushed for only a process. Many of you were at that congress and I trust that you also agree that we left some of *Alaska's business unfinished.*

I intend to share with you what I perceive to be our present state of NON-POLICY and to give you some specific illustrations of what has happened and may very well continue to happen, if WE don't do something. Finally, I have a few policy suggestions which could help bring order.

First, a couple of disclaimers. From time to time, it will be necessary to mix public health and medicine - even though in this conference we are hoping to focus on public health. Second, it will be necessary to mention a particular program or agency or geographic area in order to illustrate a point. I am not trying to impugn anyone or suggest anything illegal—I just want to illustrate a point.

Let's begin by taking a quick look at the health systems that exist in Alaska today. I know of no state that has as complicated and varied a set of systems as ours both for public health and for the provision of direct care services. Such a system HAS to contribute to the confusion of who is responsible for what.

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[1] Presented at Alaska Health Congress, Alaska Public Health Association, Anchorage, May 11, 1987. Bert Hall is currently Director of Health and Human Services, Municipality of Anchorage.

We have the federal government which funds and manages the Indian Health Service, including environmental programs, direct care for Alaska natives, and a mixture of preventive health programs. Some programs are provided directly, some through contracts with the Regional Health Corporations and other providers, and some in concert with the state. Many traditional public health programs are interwoven throughout. Some are comprehensive – others are mere gestures. Other federal programs through CDC, EPA, OSHA, HHS and other federal agencies provide additional public health services, both directly and indirectly.

Then, there is the state, itself, which provides some direct care as it manages its own hospitals for the mentally ill and severely retarded, provides funds for hospitals, runs programs in specific areas like tuberculosis control, does epidemiology, runs laboratories, funds emergency medical services, provides resources for maternal and child health programs, manages resources for alcoholism, drug abuse and mental health efforts and a variety of other programs.

In jurisdictions outside of Anchorage and the North Slope, it provides most public health services, both physical and environmental. Additionally, the legislature provides a number of competitive grants as well as directly funds both capital and operational efforts to local jurisdictions, non-profit organizations or anyone else they please.

Of some surprise to many, the federal government also manages a substantial health program through the military agencies. We tend to forget that both direct care and traditional public health services are provided at dozens of military sites, some with populations far greater than many Alaska cities. The Veterans Administration claims to be a major provider through payment of client services. I was once told that they spend over $50 million a year in Alaska.

Alaska's native Regional Health Corporations are becoming a delivery system unto themselves and now provide contract management of many former IHS facilities and programs in many parts of the state.

The private hospitals along with community and non-profits are certainly a major component of the system and though virtually all of our pre-statehood hospitals were church sponsored and subsidized, we have seen an evolution which now includes several profit-making efforts. For example, Humana is already five years old.

Also in the picture should be included the health care financing programs like Medicare, Medicaid, other insurance programs, etc..

In our diverse listing, we should not overlook the involvement of local governments, especially those with health powers who tax themselves to help pay for services.

With such a complex health delivery system in one state, why would anyone wonder that there could be confusion about jurisdiction, overlap of services, duplication of effort, or maldistribution of resources?

Believe it or not, the last time anyone made a serious effort to look at Alaska's health system was in 1954. If you have not heard of it, it is called The Parren Report. If you have not read it, you should. It's a well-done, scientifically sound report which suggested that the federal government had *unfinished business in Alaska,* the Territory. The report presented a series of well-documented recommendations. Many have been totally ignored which, if followed, would have led to a much clearer role for both Alaska as it became a state and for the federal government, itself.

Let me note that its first chapter is entitled *"Alaska's Unfinished Business"* and ironically describes many of the same confusing conditions we observe today. From its recommendation section are found statements like the following:

- "that the continuing partnership with the federal government impose fewer restrictions upon Alaska's powers of conducting its local affairs;
- that Alaska acquire more effective local political instruments for public health and related public services;
- the health problems here are essentially political, administrative and fiscal;
- in health, as in many other human problems, we must help Alaskan to help themselves;
- Alaska is unfinished business."

What the Parren Report did was to suggest that the time had long since passed for the federal and territorial governments to sit down and develop policies regarding which government should do what. As statehood came, the two entities continued somewhat in harmony, each doing its own thing, both doing some and neither doing some. It is hard to believe that 33 years have passed and still many of the problems exposed in 1954 continue unabated in 1987 — the number one of which, in my opinion is the lack of a clear understanding of the proper role for the State of Alaska now and in the 90s.

You may ask WHY is there a need to have a clear state health policy — what will we gain? How will people be better served? Will the public's health be better? What's wrong with the way we do it now? Does anyone really care?

Let me explain what I mean by a clear state public health policy, which I am suggesting would clearly identify the appropriate role for the state in areas of authority, responsibility, regulation and funding.

It is not totally true that Alaska has NO public health policy. There are, in fact, several ways to interpolate

e policy. The State Constitution, for instance, clear-
dentifies public health as a basic state responsibility.
icle 7, Section 4 states "the legislature shall provide
the promotion and protection of public health."
re are dozens of state statutes, even an entire chap-
that speaks to both physical and environmental
lth concerns. There are statutes that regulate all
ds of health concerns from communicable disease
trol to air pollution, from transportation of dead
ies to atomic energy. There are statutes that au-
rize specific grant programs. And finally, one can
ays interpret some policy from the annual budget.
hat I believe is missing is the clear overall commit-
t of the state that identifies its level of importance in
lic health matters — that clearly interprets the distri-
on of health powers to local jurisdictions; that es-
ishes a fair set of criteria for the distribution of state
urces to meet public health problems and provide
lic health services.
would venture to say that most thinking Alaskans
ld agree that Alaska has a clear policy for fish and
e matters; and that state policies on land use and
ural resources, though controversial, are basically
r and understandable. Everyone knows where Alas-
tands on education and public safety. Why then
ld it not be expected that we would have the same
public health?
would like to suggest that what we have in the area of
lic health is clear non-policy. Let me site a few
mples. I mentioned earlier the phrase "health pow-
" I challenge anyone to explain clearly what that
ns in 1987! Some of us have traced the history of
lic health in Alaska and know about the first pro-
ns, the creation of the Territorial Boards of Health,
creation of the Territorial and State Health Depart-
ts, how local programs came into being, and how
e local governments obtained local health powers.
we don't really know what it means!!!!
the 60s the former Greater Anchorage Area Bor-
h residents passed a referendum and adopted the
lth powers provided for in the Mandatory Borough
, old Title 7, replaced by Title 29, Municipal Gov-
nent. The North Slope Borough residents voted
nselves health powers. But what did those elections
n? Neither jurisdiction today has complete health
ers. In Anchorage, several areas like state facilities
ections, pollution of navigable waters, food service
ections at the airport were retained by the state.
hat those two local governments did get, however,
the opportunity to tax themselves to pay for some of
services the state provided 100% for everywhere else
e state. History shows that the more money that the
l governments spent, even on embellishments, the
the state provided. You would not be surprised to
n that even though the population and program
s have risen dramatically in Anchorage over the past
six years, state support for core services has dropped each of the past five or six years.

It is also interesting to note that when we had health revenue sharing programs, some areas like the Sitka and Bristol Bay Boroughs held heath power votes, got the money, but never provided programs. The state continued 100% provision of public health services. Here is a clear non-policy regarding health powers.

I also made brief mention earlier that another measurement of policy is seen in how monies are spent. There is absolutely no mystery to me how much of the state discretionary grants have been doled out by the legislature in recent years—certainly, not by policy but by politics. I've always prided myself that I got a fair share for the programs I represented—but that doesn't mean that it was fair.

Without even the guideline of a basic policy, it is just too easy for legislators to distribute dollars on just a political or geographic basis. There are many illustrations. Look for instance at how Manauliiq has received state health dollars in recent years. Specific dollar amounts identified by special line items for traditional programs are then lumped together as a block grant as a separate BRU—Budget Request Unit. In other words, they get guaranteed, earmarked funding while most of the rest of the areas of the state have to compete among themselves for the rest—in many cases obtaining far less per capita. That may be good politics, but it's bad policy. In fact, when that process began several years ago and there was administrative resistance, legislative intent was printed at the bottom of each appropriation to the effect that if Manauliiq was not awarded its funds as indicated, then none of the entire appropriation could be expended. Good politics—bad policy!!

Let me give you another illustration, this time a capital project in Anchorage. In 1981, one of our big capital project years, our local blood bank organization hired a well-known, powerful lobbyist whom they paid $300 a day, over $50,000 for the session. It was his job to get funds for a new building. Throughout the session, there was no public mention of the project. No local government endorsement was given nor even requested. No health planning body knew of the need or even the desire. As the house and senate budgets passed, it was not included. But when the free conference committee came out with the final document, presto, a line item of something like $2.6 million to the municipality of Anchorage was present for a new Blood Bank. So, the Municipality of Anchorage applied for the funds, passed them on to the Blood Bank group, a new building was constructed, and the lobbyist got another year's contract. Good politics. Bad non-policy.

It's my guess that you probably know as many similar stories as I do—and I think I know a lot. But suffice it to observe that a clear state policy for distributing state health resources would lead to a fairer distribution of those resources.

Let me try another approach and give you an illustration of how a clear state policy relating to statistics and the adequate collection of data could help public health programs. Supposing that we had a policy leading to a reliable data system that could be used to identify problems, prioritize them, and be useful in evaluating any success of programs developed in response. Was not this hope the gist of Susan Addiss' message to us a few Congresses back? Wouldn't a policy of good data collecting lead to another policy of investing state resources where there are proven needs and into programs shown to be cost effective?

We know that the lack of a good national data system has led to the non-funding of many worthwhile programs. Caused first by the Paperwork Reduction Act in the Carter Administration and enhanced by the elimination of over 50 major data collection systems in the present administration, it has deprived agencies and programs of convincing proof that some really work and others actually save dollars in the long run. In Alaska, most health scientists agree that our record keeping "stinks." Recent work done by the state's Epidemiology office revealed great shortcomings in existing data on deaths. There are some who even wonder aloud why public health vital statistic programs are administratively handled by coroners offices in the court system!!!

At the outset, I quoted the Daily News story on the budget and recited a litany of special interest groups and how effective lobbying saved their day. It may sound like I'm talking out of both sides of my mouth when I suggest that we too, can accomplish some of the same with the same methods. What I am really trying to suggest is that the only way we will achieve the honorable goal of a clear state public health policy is for us to develop an adequate and acceptable process and then sell it politically. The process we design must be geared toward influencing key decision makers in both the legislative and executive branches. The time is now.

This then, is our challenge. I conclude as I began:

In my opinion, there could have been no better theme chosen for this year's Health Congress - TOWARD A STATE PUBLIC HEALTH POLICY FOR THE NINTIES—ALASKA'S UNFINISHED BUSINESS. For those of use who claim to work in the field of public health or for the health of the public in Alaska, there can be no more important agenda for the next few years than for us to get involved with and influence the development of the process leading toward that goal.

How to Develop Health Policy

by Kristine Gebbie[1]

If you were to come to Oregon and look, you would t find a very coherent, visible public health policy. is is partly because we have too many actors:

- the Health Division;
- the State Health Planning & Development Agency (SHPDA);
- the Department of Environmental Quality; and
- local Health Departments,

:h with separate statutory authority to plan.

We have not always coordinated well—but we are ting better. The most recent health plan published by IPDA includes major emphasis on Maternal Child alth, and portions are lifted almost verbatim from the alth Division's 1990's objectives. We have an Inter- ency Hazard Communication Council to work on oblems associated with toxics. We do have a reasonably nerent Health Division policy, based in a Mission atement and 1990 Objectives. There are also some osets of health policy which are statewide:

- Drinking Water
- Trauma
- AIDS

ocess for Developing a Policy Process

A key decision which must be made early is the target the policy. Is it a policy for:

- the public health association,
- the official public health agency,
- all publicly funded agencies,
- all health/illness providers, or
- all Alaskans.

The broader the target of the policy, the more mplex the issues of participation, ownership and def- tion of goals. Although it is more complex to develop, recommendation is to move toward a policy for all zens, with a broad ownership.

A second major decision is the focus of the policy—is o be a policy of services to be available, or an outcome licy, or a mix? It would be possible to have a process licy, such as "prenatal care will be available, at no st, within 30 minutes travelling time, to any woman o becomes pregnant." The unstated expectation is at establishment of such a service network will be ficient to connect pregnant women with service and d to reduction in infant mortality and low birth- ight. The connection, however, is not automatic, and there are many examples of high frustration generated when a service availability goal is fully met and the problem does not evaporate.

Likewise, an outcome goal such as "achieve 100% immunization at school entry" can be frustrating if no attention is paid to the service system which must be in place to achieve the goal. People may think that a school entry vaccine policy will automatically eliminate vaccine-preventable disease outbreaks, but if there is a large unimmunized older school-age or adult population, major outbreaks can continue for a number of years and can lead to a sense of failure.

My firm recommendation is for a policy statement which is a mix. If the policy for all citizens can be stated as health outcomes (rates of diseases or injuries), the policies for specific units (official agencies or clinical services) can be developed to describe the service components which must be in place to accomplish the desired outcome.

How to Proceed

The closest thing we have to a national health policy statement is the future envisioned in the *Objectives for 1990* published by the U.S. Public Health Service. A quick look at that material illustrates some of the "How" problems in policy development.

The process was "owned" only by the agency which wrote it. Most persons in state and local health agencies were presented with a completed policy statement, first in the *Healthy People* document and then in the specific *Objectives for 1990.* While much of the material is consistent with the general goals of the public health community, there was no opportunity to consider whether the right configuration of programs was presented, or to debate the achievability of the goals. There is no way that the total U.S. minority infant mortality rate will drop unless the individual states' minority infant mortality rates drop, and the states did not share in goal development.

Taken to the state level, the same thing happened. In a number of states, the state's goals for 1990 were developed by taking the federal goals and applying them to the state demographics. This process did not allow for critique of the content areas or the goals cited. In Oregon, though we have used the 1990 Objectives to focus our state health division budget, the local health departments felt outside the process—many have not found them terribly useful. Our process for the new century will be different—and will start far enough ahead (I hope!) to allow local participation.

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esented at Alaska Health Congress, Alaska Public Health Associa- n, Anchorage, May 11, 1987. Oregon Health Division, Depart- ent of Human Resources, 1400 S.W. 5th Ave., Portland, OR 97201.

The objectives seem to be organized by the federal organizational chart. Why do we have separate sections on "maternal and child health" and "family planning?" Are "sexually transmitted diseases" distinctly different from "other infectious conditions?" Why aren't there similar separations provided for various types or causes of injuries? If there are reasons beyond the organizational chart, they have not been communicated effectively so that the logic can also support work at the state and local level. Again, in Oregon, we have regrouped several categories which made sense. As we move toward objectives for the new century, we may find some entirely new organizational system.

Any policy process must have authority for its activities. A major flaw in the policy process initiated under the federal health planning process is that it had no direct tie to the long term authority of state governments (and none at all to local government), and the sanctions involved all focused on the illness treatment system. The policies developed had their greatest impact in the illness treatment system, the plans printed often gathered dust once distributed, and there has been little effort to continue the system absent the federal authority. To legitimize a statewide health policy process, there must be a clear tie to state law or state agency authority. Occasionally a voluntary group can co-opt the decision-making process to promote a broad policy from the outside (i.e., Mary Lasker and others and the national policy on cancer research), but few groups in public health have sufficient social power to do so.

The authority of the official public health agency can provide the legitimizing umbrella for state public health policy, and therefore, should be brought in early in the process. Do not ever underestimate the convening power of the state health agency. But be sure there is something of substance on the agenda when people arrive.

Because public health activity in states is generally spread across several agencies (public health, natural resources, workers compensation, mental health), an early decision will have to be made: to define public health as circumscribed by the public health agency, or as fully defined in public health theory. If the broader definition is chosen, extensive constituency building activities will be needed to bring those other agencies and their associated interest groups into the process.

Various portions of the agenda may move faster than others. Three examples of varying rates of progress within Oregon are:

1) Healthy Mothers/Healthy Babies Coalition -
   - This is a "natural" subject area for public health.
   - There is a national logo and support system which attracts groups.
   - Extensive data are available, and it is possible to move on a county-specific agenda.

2) Trauma/Injury Prevention
   - This is a leading cause of years of potential life lost.
   - There is a constituency new to public health, with much energy.
   - A data base is being developed and accepted.
   - Support for our prevention agenda is, as yet, weak or missing.

3) Toxic Substances
   - Public health is seen as interloper into an "environmental" area.
   - Information available about chemicals is not well related to available data on human health.
   - Our "put things in perspective" view is seen as uncaring by those who react equally to any measurable chemical.

Constituency Building

If you have decided to prepare a health policy for the whole state, using a broad definition of "health," information systems become critical. You must attract to the process representatives of agencies, associations and citizen's groups who need to "own" the policy statement in order to assure action to implement it. Facts about the demographics of a population and about mortality must be published in such a way as to be useful for constituency building. Selection of specific areas which serve to interest key constituencies can help shape their interest and the debate. Among our publications have been:

- Drug-related deaths
- Teen suicide
- Method of payment for maternity service
- Indoor Air
- Trauma Registry reports

We are just starting a new follow-up on tobacco-related deaths in order to highlight this issue.

Don't make the process too global and abstract. If the policy does not relate to identifiable "hunks" of experience and identify achievable steps, the constituency you have built will drift away before you've gotten anywhere. Be very cautious about terminology—we are constantly bombarded by "health" proposals which are nothing but improvements in methods of providing or paying for illness care. Use words carefully to be both inclusive and accurate.

It may well be that within a general goal statement the process will subdivide into several subgroups moving at differing speeds toward subgoals, as noted above. There is some risk that interest will wane as time passes. The visible commitment of key participants to act on policy recommendations early in the process can give impetus to the process and validate the utility of the effort. This can include quick revisions of program regulations or funding priorities, choice of terminology in publications, or attendance at related meetings or activities.

Beginning

Almost any policy text book can give you the specific steps for a community-based policy process:

- appoint a steering committee of key, powerful people,
- draft a general policy statement,
- distribute it to a broad cut of groups for reaction/refinement,
- collate those responses and revise final statement,
- publish the policy with fanfare,
- expect people to act on it.

Without any further lecturing, this audience could probably write the invitation list for both the steering committee and the review groups, and write the follow-up instructions on how to help people implement the policy.

What I want to underscore is the time and effort involved, and some warnings:

1) Provide for staff time—someone responsible for keeping things moving, watching deadlines. The official health agency can do this, and it assures their support.
2) Mix people currently responsible with outsiders so the plan will reflect both vision beyond the present and the reality of activities.
3) Don't let the form dominate the process—if some subgroup or piece "takes off," let it go. Let the outcome be more important than the steps, even if you don't get the elegant publication you wanted.
4) Be prepared to give away ownership—where it promotes the desired outcome, some other group may be a more appropriate leader.
5) Remember you can shape the discussion even if you can't control outcome. Budget provides example.

As a final warning: it is critical to keep separate one's goal in support of public health and one's goal in a specific organization. Several years ago in Oregon, I astonished some directors of local public health agencies by stating that I gave priority to support for the health of Oregonians over support for the well-being of local health departments. It is easy to revert to defense of your unit. It is frightening to push for changes which may put you out of business.

My experience has been that there is support for those who put community good first. If we do, public health agencies will continue.

The Energetics of Swimming, Running, and Walking in Exercise Prescriptions

by Jay Caldwell, M.D., M.P.H., F.A.C.S.M.[1]

It is becoming commonplace for physicians to broach the subject of exercise with their patients. I suspect that there would be even greater interest if physicians better understood how to quantitate their prescriptions.

The single most important issue to address is the purpose of the exercise prescriptions. All else follows from that determination. Exercise for rehabilitation of an injury differs from exercise for cardiovascular conditioning, which in turn differs from exercise for weight loss. In this article I will concentrate on considerations for weight loss exercise.

You can lose weight by any exercise if you keep at it long enough, but aerobic exercise has been shown to be the safest and most effective. In aerobic exercise you use large muscle groups, non-stop, for extended periods of time. The best examples are walking, cycling, running, cross-country skiing, rowing, and certain types of aerobics and circuit training (1). Swimming is a good aerobic activity, but as I'll show, is not usually very effective for weight loss.

Since weight loss exercise should be aerobic, it can be of low intensity, which means easy. The trade-off is that since fat isn't easy to jettison, you have to keep at it. No less than 30 minutes per session, and six days a week would be best and is safe if done prudently.

Swimming is a fine all-round conditioner and toner, and when you consider its low injury potential, it's perhaps the best of the aerobic activities. But it just doesn't work well when it comes to knocking off fat deposits. There are other drawbacks too. You need a pool, and you have to know how. To understand why swimming is so ineffective for fat loss, it is important to consider the physics and physiology of exercise.

Energy, measured in calories, is consumed by muscles as they generate force. The greater the effort, the more the energy used. If you eat more calories than you use up, over a long period of time you will store those calories in your insulation material: fat.

Most of the energy that we use ends up "wasted" as friction, created when tissues slide past one another. It appears as heat that requires even more energy to dissipate. The most efficient aerobic activity, cycling, "wastes" 75% of the energy used (2). By "efficient," I mean in the mechanical sense of getting the most work done for the least amount of energy used. For successful weight loss, the most "effective" exercise is the least "efficient."

During walking or running, energy is required to overcome gravity. The amount of energy required is determined by the number of muscle contractions, not by the speed of those contractions. The implication for exercise and fat loss is that what determines how many calories you burn is how long or far you exercise, not how fast.

There is little change in efficiency as running speed increases, so whether you jog three miles in a half-hour, or dash those same three miles off in 15 minutes, the calorie cost is about the same: one calorie per kilogram per kilometer which means for a 150 pounder, about 115 calories per mile (2). The heavier you are, the more calories you burn. Although it seems a little silly to do it, carrying handweights does add a smidgen to the energy requirements.

Walking differs from running in that it is very efficient when done slowly, and therefore not very effective for weight loss. At a pace under 20 minutes per mile the calorie cost is about half that of running, or about 55 calories per mile. As walking speed increases so does the energy cost until you reach about a 13 minute per mile pace, when running becomes easier (2).

During cycling, effort is expended turning the pedals. Although body weight, gear ratios, tire size and firmness, and bike weight, design, and maintenance all enter into the equation, the energy principle is the same as it is with running. Where there is no wind resistance, such as on an exercycle, the amount of energy you use is determined by the number of pedal revolutions, not by your speed. Although admittedly inexact, the distance ratio between running and cycling for the same energy expense is about 1:3 (2).

Overcoming wind resistance also requires energy. There is negligible wind resistance in running, but it is an important contributor to the energy cost of cycling. At running speeds of less than six minutes per mile, wind resistance saps barely 1% of your energy, and even at a world record pace, the wind adds a mere 10% to the effort. On a bicycle though, the wind becomes a true foe. At about 24 miles per hour half of the effort is spent just to overpower the wind; at 29 mph fighting the wind is over 90% of the effort (2).

Streamlining and drafting are very important to the racing cyclist because the amount of body surface breaking the wind determines how much drag is created. Furthermore, since a large and presumably stronger

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cyclist can fight the wind more effectively while still presenting only a slightly larger frontal profile than a smaller rider, big cyclists have an advantage.

Distance runners, on the other hand, must overcome gravity, not the wind, so the smaller and lighter they are the better off they'll be. Except in short races and on windy days, rarely do drafting or streamlining provide an advantage.

In summary then, jogging and running, cycling, and casual walking contribute to weight loss in direct proportion to their duration, not their intensity. Walking fast or hiking, however, are always more effective than slow walking.

Swimming, on the other hand, presents a far more complex picture because skill, which translates as efficiency, has to be factored into the energy equation (3). In swimming, force is required to overcome water resistance. Although a small amount of effort is needed by some people to counter gravity and stay afloat, in swimming calorie consumption is determined almost completely by the cost of overcoming fluid drag (4).

Body form, grace, and speed are the primary determinants of drag. The greater the body surface, the more the drag. Body position as well as body composition are involved here which explains why women are about a third more efficient in the water than men. Women's fat tends to be concentrated in the hips and thighs, and since women's legs are shorter and less muscular than men's, it is easier for them to remain in a horizontal, more hydrodynamic position. In general women are fatter, too, so they float higher, creating less drag (5,6).

Although body shape affects drag, velocity determines it. Drag increases as the square of the velocity. If you swim three times faster than another person, you are fighting nine times the drag. Swimming slowly is one of the most relaxing pastimes known; swimming fast is hard; swimming very fast would be impossible were the swimmer not able to hydroplane.

Another component of the swimmer's energy equation is efficiency. Efficiency confuses the issue in swimming, but not in running, for at best there is a 5% difference in efficiency between elite runners and desk jockeys. Measured differences between average and elite swimmers reaches 30–50% (7).

As skill improves, speed and, concomitantly, drag increase; at the same time body position improves so that streamlining occurs allowing drag to decrease (8). For lap swimmers at paces between 1:30 and 3:45 minutes per 100 yards (4 lengths) the energy cost is independent of speed and body weight and amounts to 20–25 calories per 100 yards (350–475 calories per mile) (9).

The energy cost of the four basic stroke differs greatly at any given speed because the action of the arms and legs creates such a variety of efficiencies. In a study of elite male swimmers, who generally have about 40–50% higher efficiency than lap swimmers, at a slow 100 yard pace of about 1:54 minutes (100 meters in 2:00) the four strokes had energy costs, measured as calories per mile as follows: crawl (240), backstroke (250), breaststroke (400), and butterfly (500) (10,11).

Once the swimmer moves faster than a pace of 90 seconds for a hundred yards, the effect of drag outweighs any savings in efficiency. When the elite swimmer picks up his pace to about a minute per hundred, the energy cost rises to over 600 calories per mile (7).

The message seems to be clear. For the recreational swimmer who cannot sustain a pace faster than 90 seconds per hundred yards, calorie consumption will be determined primarily by the distance swum. Men should figure about 25 calories per hundred yards, women about 30% less. To swim the caloric equivalent of a 3-mile jog at that pace requires about 1,400 yards for men, a mile for women.

Improved swimming technique with a concomitant fall in drag will not result in increased calories per lap until the 100 yard pace drops below 1:30 minutes. At a pace of about 1:10 minutes the calorie consumption will approach 30 (20–24 for women) per hundred yards. It makes much more sense to swim your workout as fast short bursts.

So we're back to swimming's failure as a weight control modality. It is simply a matter of physics. A serious lap swimmer doing a set of 25 100s at a 1:20 pace will burn off up to 750 calories. A competitive swimmer over the course of a 4,000 yard workout will consume between 1,000 and 1,500 calories.

Overweight people, on the other hand, often aren't able or willing to swim fast enough for it to do any good. They are so slow they don't have enough time to do enough anyway. Many people have the sense that just getting from one end of the pool to the other is a tremendous achievement. It may be, but not in terms of fat loss. Flailing around in the water doesn't accomplish much, nor does a stately cruise.

For a woman, a pleasant 20 lengths of breaststroke may improve fitness, but will net only about 50 calories above that required to maintain flotation. At 3,500 calories per pound, your patients won't lose fat very expeditiously. So unless they are willing to learn to swim fast, it makes more sense to have your patients spend their time walking. At least they'll be guaranteed a half a calorie per kilogram per kilometer no matter what their speed, and double that if they jog.

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A/Brazil/11/78 Influenza Outbreak, St. Paul Island, Alaska

by Thad L. Woodard, M.D.[1]

ABSTRACT

An outbreak of A/Brazil/11/78 (H1N1) influenza occurred in late December 1978 and early January 1979 on St. Paul Island, Alaska. On the basis of virus isolation and/or serologic results, 106 of 203 (52.2%) individuals were considered confirmed cases. However, the attack rate was 71.6% for individuals $\leq$25 years of age and only 14.5% for those $>$25 years of age ($p<0.00001$). The predominant symptoms of this mild illness were fever (85.5%), cough (85.5%), headache (71.1%), fatigue/malaise (68.7%), and rhinitis (65.1%). No serious complications were reported.

Analysis of patients' sera for hemagglutination inhibition (HI) antibodies revealed similar rates of seroconversion against three H1N1 antigens: A/USSR/92/77 (43.7%), A/California/10/78 (40.6%), and A/Brazil/11/78 (45.2%). Surprisingly high acute geometric-mean HI titers to these antigens, especially A/USSR/92/77, for well persons $\leq$25 years of age provided evidence of unrecognized H1N1 influenza on the island within the past few years.

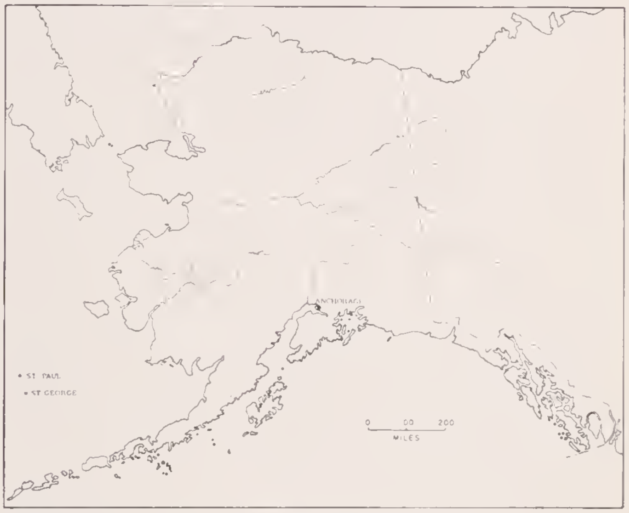

Figure 1. Map of Alaska indicating location of St. Paul Island.

INTRODUCTION

On Thursday, December 28, 1978, the Indian Health Service (IHS) physician stationed on St. Paul Island, Alaska, reported via satellite telephone an outbreak of influenza-like illness affecting predominantly children and young adults. This report was discussed by Center for Disease Control (CDC) epidemiologists stationed in Alaska and CDC virologists in Atlanta, Georgia. Because influenza had not yet been reported in Alaska that season, and because of the advantages of epidemiologic studies in isolated areas and the great national interest in influenza, a full investigation was initiated. The results of the investigation are reported in this paper.

Materials and Methods

Study population. St. Paul Island, a classic "virgin soil" island, is one of two inhabited Pribilof Islands located in the Bering Sea 200 miles north of the Aleutian Chain and 950 miles west of Anchorage (Figure 1). St. Paul, the only community on St. Paul Island, had a permanent, predominantly Aleut population of 524. Two Weather Bureau employees and 26 Coast Guard personnel lived outside town near the island's airstrip. The primary transportation between the islands and mainland Alaska in winter was by a commercial airline that had in 1978 a flight each Wednesday from Anchorage via Cold Bay in the Aleutians.

A small charter airline also provided a flight each week from Cold Bay via St. George, St. Paul's sister island. Occasional Coast Guard aircraft, charter flights, or ships and fishing vessels provided the only other opportunity for humans to introduce illness to the island. St. Paul Island serves as a bird and fur seal rookery in summer, although these animals are infrequently seen in winter. Health care on the island was provided by a single IHS physician and two community health aides. Patients were seen in a clinic adjacent to a four room hospital, all of which was physically attached to the physician's quarters.

General methods. Before the CDC epidemiologist arrived, the IHS physician obtained swabs for throat culture for virus isolation and acute phase serum from patients he was seeing and administered a short questionnaire designed to ascertain date of illness onset, signs, and symptoms. Following the epidemiologist's arrival on January 3, 1979, anyone wishing to participate in the study, whether ill or not, was asked to come to the clinic where an acute-phase serum specimen and a swab for throat culture for virus isolation, when appropriate, were obtained. Also, a more thorough questionnaire requesting information about recent illness and possible travel from the island was completed for these individuals. Convalescent-phase serum specimens were obtained by the IHS physician 4-6 weeks after acute-phase serum specimens were taken.

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To aid in data analysis, a community census involving the clinic's patient record file was taken (records were available for everyone on the island except the Coast Guard personnel).

Questionnaires. Questionnaires were administered to 65 individuals by the IHS physician and to 145 individuals by the CDC epidemiologist. Because of occasional overlap, 203 persons completed questionnaires.

Virus isolation. The IHS physician used only Culturettes®* to obtain swabs for throat culture from 37 individuals. The epidemiologist obtained such swabs from 71 individuals using standard cotton swabs that were then inoculated into viral transport media. The Culturettes® and the viral transport media were immediately frozen at -60° C and later forwarded to the Virology-Rabies Unit of the Alaska Department of Health and Social Services in Fairbanks. At this facility aliquots of the samples were inoculated into each of two tubes of rhesus monkey kidney cell tissue cultures, and the presence of virus was determined in tests for hemadsorption with guinea pig red blood cells (1). Virus isolates were identified by hemagglutination inhibition (HI) with antisera prepared against A/USSR/90/77 (H1N1), A/Texas/1/77 (H3N2), and A/Victoria/3/75/ (H3N2). All isolates were confirmed by the WHO Collaborating Center for influenza at the CDC as A/Brazil/11/78 (H1N1), a variant of A/USSR/90/77 virus.

Serology. One hundred ninety-six acute- and convalescent-phase serum specimens were obtained and stored at -60° C before being shipped to CDC. At CDC the specimens were tested for HI antibodies to the following H1N1 viruses: A/USSR/92/77, A/California/10/78, and A/Brazil/11/78. The method used was described earlier (2).

Case definitions. A confirmed case was defined as an isolate of A/Brazil/11/78 from a throat culture or a fourfold hemagglutination titer rise to any of three H1N1 antigens A/USSR/92/77, A/California/10/78, or A/Brazil/11/78. A possible case was at least three of the following symptoms: fever, headache, cough, fatigue/malaise, and rhinitis without acute- or convalescent-phase serum specimens being available for testing. Possible cases were used only in plotting the epidemic curve; only confirmed cases were used in other analyses.

RESULTS

Characteristics of sample population. The population sampled represented 38.7% of the island's permanent population (for purposes of this report, Weather Bureau and Coast Guard personnel are excluded). The age and sex characteristics of the sample varied slightly from those of the total village population. Sixty-six percent of those sampled were ≤25 years of age, whereas 55.5% of the overall village population were. Furthermore, 37.4% of the sample were males, but 51.3% of the village were actually males.

Epidemiology. Figure 2 depicts the epidemic curve for 108 cases (confirmed and possible) for which the date of onset of illness could be determined and indicates dates on which humans could have introduced illness onto the island. The first confirmed case occurred on December 21, the day following a flight from Anchorage. This flight carried a number of high school students returning to St. Paul for Christmas from several schools elsewhere in Alaska. However, none of the persons with early confirmed cases either arrived on this flight or were household contacts of people arriving on the flight, which suggested that this was not the source of illness. However, the flight that had arrived on December 13 had carried a woman who had the onset of an influenza-like illness in Anchorage on December 12. Although she refused to have convalescent-phase serum tested, the details of her illness, coupled with the epidemic curve and known incubation period of influenza, provide circumstantial evidence that she was a likely source for the

Table 1. Age-specific attack rate of confirmed cases during the December 1978-January 1979 A/Brazil/11/78 influenza outbreak, St. Paul, Alaska.

Age group (years)	Number in sample (percentage of total population)	Number Ill	Attack rate (%)
0-4	8(14.0)	6	75.0
5-9	34(61.8)	27	79.4
10-14	46(73.0)	33	71.7
15-19	24(35.8)	19	79.2
20-24	19(45.2)	9	47.4
25-29	15(36.6)	6	40.0
30-34	17(39.5)	5	29.4
35-39	11(42.3)	1	9.1
40-44	9(32.1)	0	0
45-49	9(42.9)	0	0
50-54	2(11.1)	0	0
55-59	5(23.8)	0	0
60-64	2(10.5)	0	0
65-69	2(13.3)	0	0
TOTAL:	203(38.7)	106	52.2
≤25:	134(46.0)	96	71.6
>25:	69(29.6)	10	14.5

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*Registered Trademark, Marion Scientific, Parkway, Kansas. (Use of trade names is for identification only and does not imply endorsement by CDC, the U.S. Public Health Service, or the Alaska Division of Public Health.)

illness. The scope of transmission of the illness was probably broadened by a large fire in the school on Christmas Eve that cause many individuals in the village to gather.

Incidence of influenza. On the basis of virus isolates and results of serologic testing, 106 of 203 (52.2%) individuals were considered to have confirmed cases.

The diagnosis was based on isolating A/Brazil/11/78 and on positive serologic results in 24 cases, on serologic results alone in 75 cases, and on culture results alone in 7 cases. Examination of age-specific attack rates for confirmed cases (Table 1) reveals a predilection for the younger age groups. The attack rate for individuals ≤ 25 years old was 71.6%, compared with 14.5% for the >25 age group (Chi square = 20.78, $p = <0.00001$). No illness was documented for anyone >39 years of age. Attack rates by sex were similar, 51.3% for males and 52.7% for females.

Questionnaire Response. The frequency of symptoms for 83 persons with confirmed cases is shown in Table 2. The mean number of symptoms reported was 5.3 (range 2-11). Fever, cough, headache, fatigue/malaise, and rhinitis were most common, with at least 97.3% of the persons with confirmed cases having these symptoms. Fever, headache, and rhinitis occurred with a similar high frequency in each age group. However, cough tended to decrease with age, and fatigue/malaise tended to increase. Of interest, 53.0% of the persons with cases had dizziness, nausea and/or vomiting affecting each age group, and diarrhea was noted for the ≤ 4 year age group only.

An attempt was made to evaluate vaccine efficacy; however, complete data were available for only 13 of 56 individuals vaccinated with the 1978-79 influenza vaccine, which included the A/Brazil/11/78 strains. Of these 13, four were ≤ 25, and in each case only one dose of vaccine, instead of the two recommended, was given in mid-November. All four were confirmed cases. One was asymptomatic, and the mean number of symptoms for the remainder was 3.0. The other nine vaccinees were >25, were appropriately vaccinated, and did not have cases.

Virus isolation. Thirty-one of 108 throat cultures were positive. These included 18 of 37 using Culturettes® and 13 of 71 using cotton swabs and viral transport media. Positive cultures were obtained within a matter of hours to 6 days (mean 2.5 days) after the onset of illness.

Serology. Table 3 summarizes the number and percentage of individuals with fourfold or greater HI titer change to each of the H1N1 antigens tested. The A/Brazil/11/78 antigen yielded positive results slightly more often than did the A/USSR or A/California antigens. When a fourfold or greater rise in HI titer from acute- to convalescent-phase serum was used as the criterion for a positive test, results for all three antigens correlated for 85.0% of the samples.

The acute geometric-mean HI titers of persons with confirmed cases and of well persons (possible cases are not considered) by age group were also examined for each antigen (Table 4). As expected, the HI titers measured in the acute-phase serum of persons with con-

Table 2. Frequency of symptoms by absolute number with percentage in parentheses for 83 confirmed cases during the December 1978-January 1979 A/Brazil/11/78 influenza outbreak at St. Paul, Alaska

	Age Group (years)				
Symptom	≤4	5-14	15-25	>25	Total
Fever	6(100)	34(82.9)	21(87.5)	10(83.3)	71(85.5)
Cough	5(83.3)	39(95.1)	20(83.3)	7(58.3)	71(85.5)
Headache	+	32(78.0)	18(75.0)	9(75.0)	59(71.1)
Fatigue/Malaise	+	29(70.7)	18(75.0)	10(83.3)	57(68.7)
Rhinitis	4(66.7)	27(65.9)	17(70.8)	6(50.0)	54(65.1)
Dizziness	+	24(58.5)	15(62.5)	5(41.7)	44(53.0)
Sore Throat	+	18(43.9)	14(58.3)	5(41.7)	37(44.6)
Myalgia	+	15(36.6)	13(54.2)	5(41.7)	33(39.7)
Fever ++	4(66.7)	13(31.7)	7(29.2)	1(8.3)	25(30.1)
Pain with eye movement	+	9(21.9)	9(37.5)	4(33.3)	22(26.5)
Nausea, vomiting	2(33.3)	5(12.2)	2(8.3)	3(25.0)	12(14.5)
Diarrhea	3(50.0)	0	0	0	3(3.6)
Earache	+	1(2.4)	1(4.2)	1(8.3)	3(3.6)

+ Could not be determined for this age group
++ Temperature of ≥ 100.8° F

Table 3. Frequency of seroconversion to each of three H1N1 antigens during the December 1978–January 1979 A/Brazil/11/78 influenza outbreak at St. Paul, Alaska

Antigen	Number with 4-fold titer change (%)
A/USSR/92/77	86 (43.7)
A/California/10/78	80 (40.6)
A/Brazil/11/78	89 (45.2)

firmed cases were uniformly low, irrespective of age or antigen, although the values tended to be lower for the 25 age group. Well persons ≤ 25 had HI titers that were significantly ($p < 0.001$) higher than those of persons with cases in the same age group, reflecting previous H1N1 exposure. Surprisingly, the acute geometric-mean HI titers of well persons < 25 years of age were also significantly ($p < 0.001$) higher than those of persons with cases in this age group.

DISCUSSION

The respiratory illness that occurred in late December 1978 and early January 1979 on St. Paul Island, Alaska was caused by the A/Brazil/11/78 strain of H1N1 influenza virus. The virus appears to have been introduced by a human in mid-December and to have caused an outbreak of illness that peaked at the end of December and subsided early in January. Individuals ≤ 25 years of age had a high attack rate, 71.6%. Older individuals had a considerable lower attack rate, 14.5%. Both groups also had high acute-phase titers to three H1N1 antigens, indicating earlier exposure to a virus or viruses antigenically similar to A/Brazil/11/78. The illness was mild and caused no mortality or serious complications.

This description is characteristic of H1H1 outbreaks (4) that have caused mild illness primarily for individuals < 25 years of age. The dramatic attack rate for the ≤ 25 year age group has been noted for relatively closed populations such as persons at military bases and schools (4,5). Because the illness was mild, the attack rate for older individuals was relatively low, and people tended to stay in family groups for the holidays, the total impact on the community was low. After this outbreak, A/Brazil/11/78 infection spread throughout Alaska.

A benefit of the study was the information provided by the descriptive epidemiology based on a large number of culture-proven or serologically proven cases. It was unfortunate that the vaccine efficacy could not be better evaluated. Although data were limited, results suggest that a single dose of the 1978–1979 vaccine did not prevent infection but may have ameliorated the illness slightly for individuals ≤ 25 years of age. Reasons for the absence of a protective effect of the vaccine include the inadequate antibody response stimulated by a single injection and the slight antigenic drift of the infecting virus relative to the virus strain in the vaccine.

High acute-phase HI titers of well persons ≤ 25 years of age suggests that there were unrecognized H1N1 infections in this age group. Of 38 individuals in this category, 27 had acute-phase titers of at least one of the three H1N1 antigens tested. The youngest such individual was 2 years old, indicating an exposure within the past 2 years.

Table 4. Acute geometric-mean HI* titer of persons with confirmed cases and well persons by age group during the December 1978–January 1979 A/Brazil/11/78 influenza outbreak, St. Paul, Alaska

		Geometric-mean HAI titer		
Age Group	Antigen	Ill persons	Well persons	p-Value
≤ 25	A/USSR/92/77	9.8	28.3	<0.001
	A/California/10/78	6.1	12.7	<0.001
	A/Brazil/11/78	6.9	14.7	<0.001
> 25	A/USSR/92/77	11.5	50.1	<0.001
	A/California/10/78	7.6	33.1	<0.001
	A/Brazil/11/78	10.7	54.3	<0.001
All ages	A/USSR/92/77	9.9	40.0	<0.001
	A/California/10/78	6.2	22.7	<0.001
	A/Brazil/11/78	7.2	32.5	<0.001

***HI = Hemagglutination inhibition**

The high mean acute-phase titers of well persons ≤25 years of age might reflect a cross-reaction to previous immunization with an H1N1 vaccine, the likely candidate being A/New Jersey/77 (Hswl N1). However, a chart review failed to substantiate this possibility, and the Bureau of Laboratories, CDC, Atlanta, did not note any priming effect by the swine influenza vaccine in its studies (personal communication, Dr. Gary Noble).

These findings may suggest earlier H1N1 influenza exposure for some individuals ≤25 years of age. Supporting evidence for this explanation is the elevated mean titer to the A/USSR/92/77 antigen relative to those to A/Brazil or A/California antigens in this group, which suggests earlier exposure to a virus identical to or very closely related to the A/USSR/92/77 antigen. The supposition that the avidity of the antigen varies does not explain the lack of difference in titers to the A/USSR and A/Brazil antigens of persons with cases and of well persons >25 years of age.

It is unlikely that all of a group this size acquired antibody from infection off the island, so it seems likely that they were exposed on the island. During the 1977-78 influenza season, A/USSR/90/77 (H1N1) virus and two H3N2 strains, A/Texas/1/77 and A/Victoria/3/76 (5) circulated widely in the United States. In Alaska there were isolates of all three of these influenza A isolates, but judging from the relative numbers of each, A/USSR/90/77 activity was minor. Furthermore, influenza-like activity was reported on St. Paul in April 1978 but was not investigated. Thus, the titers measured against H1N1 for individuals ≤25 years of age on St. Paul probably reflect an outbreak of influenza on the island the previous season. Presumably either or both of the H3N2 strains circulated simultaneously with A/USSR/90/77 and prevented large numbers of individuals from having H1N1 antibody.

An interesting sidelight of this investigation was the usefulness of Culturettes® in keeping virus viable during shipment to a diagnostic laboratory. The ability to freeze the samples improved the quality of the samples by controlling bacterial overgrowth. The materials needed are often readily available and provide acceptable results.

ACKNOWLEDGMENTS

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History of Medicine in Alaska

JOHN C. TOWER, M.D.

John Cramton Tower, M.D., was the oldest of the two children of a pediatrician father and a musical mother. He was born on September 6, 1925 in Meriden, Connecticut.

John remembers being disturbed by the great depression. His kindly, sensitive father, a graduate of the College of Physicians and Surgeons, Columbia University, had just begun to succeed in his practice when the carriage trade went down the tube. His father during this time was fortunate to make $200 a year.

John's mother was unique. She was intelligent, talented, and played the violin. At eighteen she chose to go to New York City rather than to college. Here she studied under Leopold Auer, an outstanding violinist and teacher. Among her many suitors was a Danish nobleman, but she preferred the young pediatrician.

As a child, John was frail, suffering from asthma, hay fever, and eczema—afflictions he finally outgrew but not without incident. His mother emphasized two things: books and music. Reading became a way of life, because not having gone to college, she desired to make up for it as much as possible. Music was natural for her. While John claims no special talent musically, he played the flute, not an easy instrument to master. His sister was a good violinist, and his father was given a clarinet. Beginning with this unusual quartet of two violins, a flute, and a clarinet, the Monday night musicale began. First growing with the addition of a Polish pianist neighbor, it eventually peaked at 32 members. The "Geraniums" group would concertize at local institutions. John remembers playing at the insane asylum, an acceptable title at the time.

These catholic interests provided a fantastic background. John attended the Meriden High School which, as many public schools during the depression, was excellent. He looks back on his secondary school days and describes himself as being lefthanded but having a fertile imagination; acquiring a huge vocabulary from his wordy family but not being able to spell; and reading dauntlessly. While repeatedly on the honor roll, John Tower does not feel that he was conspicuously outstanding. His father had worried about not sending him to a prep school, but with the combined remarkable stimulation of family and school, John felt no loss.

Being under draft age, he was able to spend two terms at Dartmouth College. He found it electrifying. However, anticipating the inevitable, he tried to get into the Navy. He was turned down because of "buck teeth." The reason given was that the Munson Lung did not work effectively with such teeth and, consequently, he could not escape from submarines. He tried the Army but with no success.

He was drafted successfully, however, and put into an intelligence company where the qualification was having an IQ higher than 140. John was sent to Macon, Georgia. It was in the heat of summer. His training appeared to be the same as for other units. A sergeant explained that the intelligence company would lead their men into battle to draw fire so that it would be known where the machine guns were.

John tried to make a change. He opted to become a truck driver but had never learned to drive. Next, he attempted the radio corps but failed to learn morse code. He was placed in an infantry brigade which was training at Hattiesburg, Mississippi. They were outdone by the Nisei battalion from Hawaii training nearby.

Then, having been transferred to the 75th Division at Breckenridge, Kentucky, his health broke down. He developed infected eczema. Rather then accept a medical discharge, he had his father send the remedy used in his childhood.

He was sent overseas with the 36th Armored Infantry Regiment, 3rd Armored Division just in time to take part in the Battle of the Bulge. His unit was part of the northern army attached to the British. They helped reduce the northern salient and broke out of the Ruhr pocket to take Cologne. John claims that he was one of the first private first class soldiers up the spire of the Cologne Cathedral.

John's regiment then drove across Europe in half tracks following the tanks. They broke through the German lines and crossed the Remagen Bridge two days after it had been captured. Continuing on, they drove one hundred miles through the German lines. At Paderborn they met German women SS troops with 88mm antiaircraft guns. Here, in crossing the airport, John received a trivial wound. A sniper hit his bazooka, the shell bounced off a grenade and grazed his hip. He received a Purple Heart.

By now the Germans were disorganized. John's unit reached the Elbe River at Dassau. Nearby was the Nordhausen concentration camp. He remembers the scarecrow figures in striped pajamas who rose from the potato furrows. One of them turned out to be a former Belgian cabinet minister who spoke faultless English and who had been wounded by the departing Germans. John carried him to a farm house demanding that he be fed. The farmers threw him out. John found this unspeakable.

The end of the war in Europe came. Serving in the Army of Occupation, John suffered an asthma attack and was evacuated to Paris and then back to the States where he was discharged.

Happily, it was back to Dartmouth where he met Betsy Bingham who characteristically was visiting the Dartmouth Outing Club. Vassar College, which she attended, had shortened its calendar so that she was graduated in three years, catching up with John. He received his undergraduate degree from Dartmouth and a diploma from its two year medical school. Betsy and John were married on September 2, 1949. Then Betsy and he attended Western Reserve Medical School, being graduated in 1951.

They both had internships and residencies at Yale New Haven Hospital, his in pediatrics, hers in internal medicine. John had three years training while Betsy took a year off to experience motherhood in the person of daughter, Christine.

John feels that coming to Alaska was predestined. His maternal grandfather practiced ear, eye, nose and throat in St. Johnsbury, Vermont. The grandfather and a friend, who was a Republican wheel horse and a crony of Calvin Coolidge, visited Alaska for three or four months in 1928. They traveled from Lake Clark to the Aleutians, thoroughly enjoying the experience. The grandfather had numerous Alaskan hunting trophies in his house. John remembers being allergic to the bear.

The grandfather, not the allergy-making bear, could have preconditioned him. One night, he woke Betsy to announce: "We're going to Alaska." To which she mumbled: "Why not Hawaii?" and went back to sleep.

He then wrote the Commissioner of Health, Dr. C. Earl Albrecht, about the possibilities of employment. He was encouraged to come since Alaska needed pediatricians, and further, Dr. Albrecht guaranteed him a consulting position with the Department of Health and the Bureau of Indian Affairs as well. Dr. Albrecht telephoned from Washington, D.C., and they met in New Haven. Any doubts about Alaska were resolved by the terrific stories told by Dr. Albrecht. To go was now inevitable.

Betsy, John and Chris drove the highway becoming more and more impressed with Alaska. Upon arrival in Anchorage, they found Helen Whaley and Adelaide Sower, a classmate from medical school, who had flown to Anchorage. They had been promised the same things by Dr. Albrecht as had John. Adelaide left, but Helen remained to join the Anchorage Medical and Surgical Clinic.

The Towers found Anchorage most congenial. They met the late Bernie Kosinski[1] at a gas station. He was from Connecticut which created a bond. Calling the Jack White Co., they learned that Dr. Palka, the first board orthopedist, had crashed into the Inlet near Indian and had drowned. They were shown his house on 2nd Avenue, a former railroad house renovated for office space. Betsy and John purchased it immediately.

John Tower hung out his shingle six days after his arrival. In this summer of 1954, a polio epidemic had just begun. He was to receive much publicity in the newspapers and on TV because of his work. The house had a commodious front hall which he used as a waiting room. He recalls that Chris was gregarious and would play in the front hall with the patients, some of whom he sent to the hospital with polio. At one time, there were 28 people in iron lungs.

Betsy served as jack-of-all-trades. Marge Freeland, a nurse, was an outstanding help. John charged $7 for an initial visit which he thought was steep. But this did not deter one family from Ninilchik with six children. His practice grew, for in addition to the polio epidemic, families such as the Odoms, Atwoods, Stevens, and Sullivans came to him. Finally, he had to cut off new patients at the arrival of the Lowell Thomases.

By then, Helen Whaley and he were in joint practice. They were to set a standard of excellence for pediatrics in Anchorage which has been maintained ever since.

Upon arrival, he became Chief of Pediatrics at Providence Hospital, a position he alternated with Helen Whaley. However, when he went to the Bureau of Indian Affairs, they had never heard of him. He simply announced that he was their new pediatric consultant. There were 75 children in the hospital at this time with no specialists. He found illnesses on the ward which he had never seen in training: Pott's Disease, tuberculous pericarditis, otogenic meningitis, and rheumatic heart disease in overwhelming quantity. Many children died of chronic diarrhea and inanition after chicken pox.

He did his own fluoroscopy and cardiology. Working with the local undertaker, Neil McKay, in the grimy basement of a dilapidated building at the corner of 4th and C under a 60 watt bulb, he performed his own autopsies.

He found the territorial days exciting. They were filled with the stimulation of the practice with Helen Whaley and later with Harvey Zartman. It was great to be in Anchorage until after the earthquake.

With the discovery of oil, Anchorage grew to be over 100,000 and lost its frontier aspects. However, there was a plus side, for the hospital improved, the medical library grew, and there were fine new associates.

In the 60s, there was new ground to till as John helped to found, with Helen Whaley, Ken Moss, and Ken Fleshman, the Alaska Chapter of the American Academy of Pediatrics. John was to serve on two committees of the American Academy of Pediatrics—Indian Affairs; and Sports Medicine, Recreation and Physical Fitness.

Until quite recently, pediatrics was burdened with the problem of premature infants. The new neonatal care has been an enormous advantage, although there is a certain disappointment at its disappearance from pediatrics to become a specialty of its own. One can be sad at this sign of progress, but glad because it was such hard work.

---

[1] Bernie Kosinski was the long time manager of the Anchorage Times, a wonderful man, who drowned in Hawaii while saving one of his children.

As a hobby, John Tower has been playing tennis and grinding refractory telescope lenses. The lenses are more for satisfaction than use since it is often too cold in the winter for star gazing, and there are no stars in the summer.

John prides himself on being a fourth generation physician. It is personally gratifying that his son, Steve, is now the fifth.

One can harbor no fears for the future of John Tower who is imbued with enormous curiosity. Alaska and Anchorage have been benefited greatly because of his grandfather's serendipitous visit to Alaska when Calvin Coolidge was president.

Gwynneth Gminder Wilson
Chairman, ASMA Auxiliary History Project

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Glimpses of Alaskan Medical History

Edited by Robert Fortuine, M.D.

Illness in a family from Norton Sound (1778)

The 1778 voyage of Captain Cook in the *Resolution* and *Discovery* needs little by way of introduction. In September of that year Cook had returned southward through the Bering Strait and sailed into Norton Sound, where the encounters described here took place on the 13th, near the present village of Shaktoolik.

Lt. James King was the astronomer on board the *Discovery* at this time, but later succeeded to the command of the *Resolution* following the death of Cook. William Ellis, who was surgeon's second mate on the *Discovery,* later achieved some notoriety when he published his own diary of the voyage before Cook's and King's official account appeared.

King was apparently the first to meet the family, with Cook and Ellis, perhaps later the same day, probably encountering them together. The father's blindness could have been due to cataracts, infection, or perhaps even trauma, since the mother was reported as trying to communicate a misfortune that had befallen them. The young man seems to have had two problems—a condition which was "eating up" his face, and a crippling condition of his extremities. Despite his problems, however, he apparently was an active individual who could handle a skin boat, although he was unable to walk. The nature of his disease(s) must remain obscure. He may have had a kind of congenital arthrogryposis (similar to Kuskokwim disease) or simply severe clubfoot deformities. He had in addition a destructive lesion of his face, such as a chronic granulomatous infection, or more likely, a malignant tumor, possibly of the nasopharynx.

[King]: "Whilst we were at the wooding place a family passd by us, & on beckoning them to land, an elderly Man & middle ag'd woman came to us....[U]pon giving the woman a small knife, & making signs that we would give larger ones for some fish, she made me to understand that if I would walk farther on to where the boat was going I might have some; I accordingly set out with this very Civil Couple, & I had walk'd a mile before I observd that my Companion was quite blind; for on crossing a stoney beech the poor fellow made a false step & cut his foot terribly; this made us stop, & the woman pointd to the mans Eyes, which I observ'd to be cover'd with a Whiteish film;... [A few hours before] a small Canoe came by, & the same motion for coming to us being Obey'd, we found its owner the most miserable object that was ever seen: He was a young lad, who had a disorder in his face which had already destroy'd one eye, & the sides were much Swell'd, & one half of his mouth & Nose in a sad condition; we were surprised at seeing such an Object with great dexterity paddling the canoe in the Surf, & so managing it that the Surf hove it upon the beech quite dry. When he step'd out, we found that his Legs were so contract'd that he was oblig'd to crawl about on his Knees and hands; he had a few fish, for which he was well paid, & went away. This poor creature we now met, seemingly taking care of the large boat;... I was now made to sit down & perform upon the old mans eyes a singular ceremony, & which the woman had some pains to make me comprehend: this was holding my breath, & after breathing upon the Eyes, I was made to spit upon them; when this was done, seemingly to their great Satisfaction, he took my two fists & prest them against his stomach, & made me continue doing so for some time; the Woman not only now, but on the road kept telling me a long story, in a very Melancholy, whining tone, pointing to her husband, her Child, and the poor deform'd fellow, seemingly to raise my compassion, & to make me sensible of some particular misfortune that attended their situation."(1)

[Ellis]: "In the forenoon, our people on shore were visited by an old woman and a man, who appeared to be her son,... The man was a most miserable spectacle, and apparently eaten up with disease."(2)

[Cook]: "The 13th In the afternoon a family of the Natives came near to the place where we were taking off wood; I know not how many there were at first, I saw no more than a Man, his wife and child and a nother man who bore the human shape and that was all, for he was the most deformed cripple I either ever saw or heard of, so much so that I could not even bear to look at him: this state he seemed to have been in from his birth...."(1)

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1. Cook, James. The journals of Captain James Cook on his voyages of discovery. Three volumes. Edited by J. C. Beaglehole. Cambridge: Hakluyt Society, 1967;3(2):1439-1442; 1967;3(1):438-439.
2. Ellis, William. An authentic narrative of a voyage performed by Captain Cook and Captain Clerke... (1782). Four volumes. Amsterdam: N. Israel/New York: Da Capo Press, 1969;2:16.

Arctic Research: What, Why, How?

Many individuals and organizations have enthusiastically endorsed the need for an arctic health science policy. In the United States, the American Public Health Association (APHA) led efforts to develop a national arctic heath science policy—published by APHA in August, 1984. The purpose of such a policy, applicable to all circumpolar nations, is "...to assure that we are sufficiently informed so that national development, environmental explorations, and national defense occur in a manner that promotes and protects the health and quality of life of people living and working in the arctic." An arctic research policy "...is a set of guidelines and principles to:

1. identify research needs,
2. develop, coordinate, sustain, and evaluate research programs, and
3. disseminate research results in order to enhance health and advance biomedical knowledge."

Of greatest importance is that an arctic research policy constitutes "...a clear statement of commitment to develop a vigorous, continuing program of arctic health research."

In the United States, numerous groups have, since the enactment and signing into law of the Arctic Research and Policy Act of 1984, been working actively to turn this historic law into a living program of health research to benefit the people of the arctic.

After great deliberation, a consensus has been reached on the major areas where future arctic research efforts should be focused. These areas have met the following criteria:

1. Research proposals must go through peer-review to insure their scientific merit.
2. Research should concentrate on areas neglected in the past.
3. There should be evidence of cooperation among agencies, organizations, and researchers.
4. There should be evidence that research conducted will benefit people—both in the arctic and elsewhere.
5. Research should be able to be accomplished with enhanced resources.

A consensus now exists among arctic health researchers and health workers as to the major areas for future research:

- Injury Control
- Cancer
- Diet, Atherosclerosis, and Cardiovascular Disease
- Infectious Disease

Detailed descriptions of these four areas that follow are reproduced from work of the Federal (U.S.A.)—State (Alaska) Task Force on Health Research, a working group of the U.S. Arctic Research Commission; co-chaired by George E. Hardy, Jr., M.D., Anne P. Lanier, M.D., and John P. Middaugh, M.D., by permission.

I. Injury Control Research

In 1985, the National Research Council and the Institute of Medicine, in a major publication, *Injury In America. A Continuing Public Health Problem,* found injuries to be the principal public health problem in America today. Calling injuries a modern "plague of the young," the Committee on Trauma Research urged new federal initiatives to understand, prevent, and treat the problem. In the United States, injuries:

- are the most costly health problem, estimated at $75-$100 billion each year,
- are the number one killer from birth to age 44,
- strike one of three Americans every year,
- caused 143,000 deaths and 70,000,000 nonfatal injuries in 1983,
- disabled 80,000 people nationally with permanent injuries to the brain or spinal cord alone, and
- caused loss of more years of working life than all forms of cancer and heart disease combined.

Research in Alaska provides a unique opportunity to respond to the challenge of the National Research Council and Institute of Medicine by addressing the research goals and objectives outlined in *Injury In America.*

In Alaska, injuries are the leading cause of death for ALL age groups and account for 37% of all deaths in Natives and 29% of all deaths in non-Natives. Injuries also are the leading cause of years of potential life lost (YPLL) in the two populations (58% and 53% respectively). Unintentional injuries account for 39% of YPLL among Natives and 41% of YPLL in non-Natives, while intentional injuries (suicide and homicide) are the second-ranked cause of YPLL; 19% in Natives and 12% in non-Natives. Alaska is a young state—89% of Alaska's population is less than 50 years of age—**injuries account for 70% of all deaths in Alaskans less than 50 years of age.**

Research in Alaska can provide statewide, population-based, accurate information on the ability of currently existing data systems to define adequately the occurrence of injury. Because these data systems exist in all states, strengths and deficiencies identified in Alaska will be similar to those that exist in other states. Improved collaboration between different federal and state agencies will result in more efficient use of resources and will avoid unnecessary duplication of efforts. Accurate information will enable focused efforts at highest risk subgroups and will identify risk factors worthy of detailed investigation.

Integration of existing data sources and data systems will result in an ongoing comprehensive statewide injury surveillance system. Ongoing evaluation of the surveillance system will identify mechanisms to enable their application to other states and to national programs. Injury research in Alaska will result in defining the spectrum of intentional and unintentional injuries using age-, sex-, and race-, and injury type-specific data and enable the detailed investigation of the contribution of injuries to mortality, potential years of life lost, morbidity, disability, and costs by injury-specific category. As reported by the U.S. Arctic Research Commission, May 30, 1986, "National Needs and Arctic Research: A Framework for Action":

> "The occurrence of voluntary and involuntary injury, suicide, homicide, and death due to alcohol are extraordinarily high in the Arctic. Research initiated in the Arctic focusing on high-risk populations will be effective in identifying intervention strategies having universal application."

II. Cancer

In the United States, cancer is the second most frequent cause of death as well as second as a cause of years of potential life lost (YPLL) before age 65. In Alaska, cancer ranks somewhat lower due to the young age of the population, but still is in the top five causes of YPLL in both Natives and non-Natives. In the Alaska Area Native Health Service, cancer is the third most frequent cause of inpatient hospital days, following only injuries and deliveries.

No data are readily available on numbers of cancer cases or extent of hospitalization in the non-Native population in Alaska. However, accurate data are available on cancer incidence in Natives. All new Native patients with cancer have been registered since 1969. When age of the population is taken into account, the rates of cancer in Natives are similar to that of United States whites. This is an astounding fact since as recently as thirty years ago, cancer was thought not to occur in Eskimos and other Natives.

Although the total cancer burden is the same overall, the types of cancer (that is, body organs in which cancer develops) differ markedly in Natives from other populations. Alaskan Natives are much more likely to develop certain types of cancer (nasopharynx, esophagus, cervix, liver, gallbladder, kidney). While some types of cancer (breast, prostate) currently occur somewhat less often in Natives, rates are increasing. Unfortunately, no types of cancer are decreasing. Data from other countries indicate cancer patterns and trends in Native Arctic residents in Canada and Greenland are similar to those in Alaskan Natives.

Cancer is a disease of persons largely over age 40. Since Native people are generally living longer and certain cancer-causing practices (e.g. tobacco use) are unusually prevalent, one can predict an increase in the numbers of cancers. In fact, in approximately 30 years, the numbers of new cancer patients diagnosed each year among Natives will be at least twice the number seen today.

Ability to screen for cancer and recommendations for screening programs may differ in the Arctic from elsewhere because the types of cancers in the Native populations differ from the United States; persons are widely dispersed in small, geographically isolated groups; and sophisticated equipment (mammography, sigmoidoscopy) needed for cancer screening and diagnosis is usually available only in the urban centers. In addition, cancer information brochures and educational materials readily available are generally not culturally applicable to Native populations.

Although much is known about cancer, much remains unknown about this disease, elsewhere, as well as in Arctic populations. Comparison of cancer patterns among various Arctic populations and between Arctic populations and others may yield clues to the causes of cancer. Similarly, examination of changes in cancer patterns over time may also yield clues. Tobacco use is thought to be responsible for at least one-third of cancers; knowledge of ways to prevent the use of these products and to reduce or eliminate the level of use in current users is desperately needed. Secondly, viruses are believed to play a key role in certain cancers—several of these viral-associated cancers occur at increased rates in Arctic Native residents. Understanding of the role of these viruses in cancer would impact Native and all other populations. Thirdly, environmental and occupational causes of cancer are always of concern. Accurate cancer incidence data are needed on Arctic residents to assess the role these factors play. Finally, genetic factors also play a role. Since Arctic families are large, tend to remain in Alaska, and accurate medical histories are available, studies of the patterns of cancer in families may help understand the role of genetic factors in cancer.

III. Diet and Atherosclerosis

Cardiovascular disease due to atherosclerosis remains the chief cause of death in the United States, Western Europe, and Alaska. Epidemiologic studies have documented a remarkable finding: the low incidence of coronary heart disease (CHD) among the Eskimos. Numerous clinical and biochemical reports and studies attribute this low incidence of coronary heart disease to omega-3-polyunsaturated fatty acids in cold water fish and marine mammals that constitute a substantial portion of Eskimo diets.

In normal adults and patients with CHD, omega-3-fatty acids have been reported to produce beneficial protective effects including:

- reductions in platelet aggregation, thrombotic tendencies, and blood viscosity;
- reductions in elevated serum cholesterol, triglycerides, cholesterol-rich LDL, and triglyceride-rich VLDL; and
- increases in low serum HDL.

The prevalence of atherosclerotic disease among Eskimos has been demonstrated to be less than that among other Northern American populations. This information, however, is based on a limited series of examinations performed three decades ago. Research must verify these earlier observations. Recent technological advances have led to dramatic breakthroughs in our understanding of lipid metabolism and the association of increased levels of high density lipids with protection from atherosclerotic disease and its complications. Extensions of present understanding of the roles of prostaglandins and thromboxanes in coronary artery vasospasm, platelet aggregation, myocardial infarction, and stroke have yet to be extended to arctic indigenous peoples. Dietary influences on prostaglandins, thromboxanes, cholesterol, and lipids associated with traditional Native food consumption offer exciting possibilities to further our knowledge.

The beneficial consequences to the nation could be sweeping if increased consumption of a renewable resource such as salmon reduced atherosclerosis and cardiovascular disease. Additional studies on atherosclerosis and heart disease need to be conducted in the arctic with particular emphasis on these critical research areas where solutions will have broad application to the United States and to other circumpolar arctic nations. Research needs include extension of clinical epidemiology, biochemistry, nutrition and diet surveys, and laboratory work.

IV. Infectious Disease

The 1990 Objectives for the Nation target numerous infectious diseases for reduction in illness and the consequences. Areas include:

- hepatitis B
- tuberculosis
- pneumonia/influenza
- bacterial meningitis
- hospital-acquired infections
- sexually transmitted disease (gonorrhea, syphilis, herpes, chlamydia, other)

Among Alaskan Natives in 1950, infectious diseases were the leading cause of death. Rates of death per 100,000 persons from infections were extraordinarily high (tuberculosis 673.3/100,000; influenza and pneumonia 192/100,000; and other acute infectious diseases 91.6/100,000)—30, 10, and 7 times as high as U.S. whites, respectively. Infections were also the leading cause of illness.

Cooperative federal-state programs were implemented to control these infectious diseases, especially tuberculosis. Since then, many combined research and health delivery programs have successfully reduced morbidity and mortality among arctic residents while contributing important research funding. Examples include extensive work on zoonotic diseases (trichinosis, diphyllobothrium, and rabies), bacterial illness (streptococcal disease, botulism, haemophilus influenzae, and meningococcal meningitis), and viral disease (hepatitis A, hepatitis B, influenza). The national importance of recent research and vaccine trials in hepatitis B and haemophilus influenzae in Alaska is well recognized.

Many opportunities exist for continued research on infectious diseases in the arctic that will be of national importance. Fruitful efforts can be expected to contribute to increased understanding and improved ability to control respiratory and diarrheal diseases, hepatitis A, botulism, and chlamydia. New diagnostic agents and vaccines now under development could lead to additional reduction in the burden of infectious diseases. Additional research in infectious diseases would have importance, not only to the people of the arctic, but to the nation and the world.

For example, pneumonia/influenza ranks sixth in the United States among leading causes of death and tenth in potential years life lost (YPLL). In addition to being the cause of many deaths, infectious diseases contribute to several-fold more deaths than they cause. Each year over 2 million pneumonias are diagnosed in the United States with 400,000 persons suffering from pneumococcal pneumonia alone at a cost of $325 million.

In Alaska, pneumonia/influenza is the ninth most frequent cause of death. As a cause of years of potential life lost (YPLL), it ranks seventh and ninth among Natives and non-Natives, respectively. In the Alaska Area Native Health Service, pneumonia is the fifth most frequent cause of hospitalization and ranks even higher in areas defined as "Arctic." Approximately one-fourth of pneumonia and one-half of otitis media are estimated to be caused by a single agent, the pneumococcus. Infections by the pneumococcus appear to occur more than 15 times more often in Native children and adults.

A research initiative focused on the causes, diagnosis, and prevention of respiratory diseases, especially pneumonia, in the arctic could yield substantial improvement in health. Further research on botulism, hepatitis A, gastrointestinal infections (including bacterial, viral, and parasitic infections), and sexually transmitted diseases (chlamydia, especially) are needed.

PATHOLOGICAL AND INFECTIOUS WASTE

Revisions of the Department of Environmental Conservation's Solid Waste Regulations 18 AAC 60 became effective September 20, 1987. These revisions include certain requirements specific to the disposal of pathological or infectious waste.

Infectious waste as defined by these regulations means, "certain laboratory, surgical, and hospital waste; surgical specimens including pathological specimens, tissues, blood elements, excreta, and secretions obtained from patients; disposable materials that have been in contact with persons who have a suspected or diagnosed communicable disease; a substance that might harbor or transmit pathogenic organisms; disposable materials from outpatient areas, emergency rooms, and rooms of patients with a suspected or diagnosed communicable disease that requires isolation; and certain equipment, including instruments, syringes, and needles."

Subsection 18 AAC 60.087(g) states: "A person who disposes of a pathological waste or an infectious waste shall first disinfect or sterilize, and then package the waste to prevent a health hazard, or shall incinerate the waste in a pathological-waste incinerator before disposal at an approved solid waste disposal facility."

For more information contact the following offices:

Department of Environmental Conservation

Northern Region
Doug Lowery
P.O. Box 1601
Fairbanks, AK 99707
452-1714

Southcentral Region
Henry Friedman
3601 C Street, Suite 1350
Anchorage, AK 99503
563-6529

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My Turn

When a physician gives a prognosis that "nothing can be done to alter the course of a disease or condition" the patient may become desperate and turn to others who promise a cure or treatment. We have seen this with the Krebiozin and Laetril scandals, and we are now seeing it with "Sicca Cell Therapy" being offered to children with Down Syndrome. This is a form of "immuno-therapy" which is being popularized by a German practitioner who presents annual seminars on the west coast of the United States. Families also travel to Germany to get the initial course of intramuscular injections of lyophilized fetal lamb or pig brain cells.

The good news and the proper prognosis regarding Down Syndrome is that early educational intervention does improve the child's prognosis for learning and does improve the parents' ability to cope with a child with a chronic handicap.

The following position statement has been developed by the Alaska Division of Public Health to communicate a sensible approach regarding this question of Down Syndrome treatment.

> The Division of Public Health has become aware of Alaskans who are utilizing "sicca cell therapy." This is an unproven therapy in which lyophilized (ground up) cells of fetal animals are administered by intramuscular injections to children with mental handicaps, particularly to children with Down Syndrome.
>
> With regard to this so-called "therapy," in December, 1985, the National Down Syndrome Congress issued an official statement which says in part: "There is serious concern that the injection into humans of animal cell preparations carries a danger of long-delayed, fatal, 'slow-virus' infections. The potential incidence of this is unknown. We feel that the use of so-called 'sicca cell' therapy should be viewed as threatening to life." The entire statement follows.
>
> The Alaska Division of Public Health, until results are available from properly designed research studies, discourages use of this unproven therapy for the following reasons:
>
> (1) There is a definite potential for harm from infection and from immune system adverse effects, as a result of injecting material that may be contaminated by "slow virus" particles;
>
> (2) There are high monetary costs for these injections, and there are significant emotional costs to parents as a result of failed expectations; and
>
> (3) It is illegal to use this "therapy" in the United States because it lacks the necessary Food and Drug Administration approval and because sending the material via mail or causing an innocent party to issue and mail a check based on fraudulent information constitutes mail fraud.
>
> Mental retardation, including Down Syndrome, can best be managed by using accepted and proven effective medical and surgical treatments and by early use of special education methods and tech
>
> Approved by:
>
> Elizabeth Ward, R.N., M.P.H.
> Director, Division of Public Health
> Department of Health and Social Services
> June 29, 1987

The Down Syndrome Congress statement made December 23, 1985 on this subject is also presented here for further information:

> Statement by the
> National Down Syndrome Congress
> on
> Sicca Cell Therapy
>
> The National Down Syndrome Congress, as the primary parent representative for those families affected by Down Syndrome, has not previously made an official statement regarding any type of proposed therapy relevant to Down syndrome.
>
> However, at this time, we would like to indicate that:
>
> (a) sicca cell therapy is not licensed by the Food and Drug Administration (FDA)
> (b) that potential or at least theoretical risks do attend the inoculation of foreign proteins and biological substances into the human body.
>
> There is serious concern that the injection into humans of animal cell preparations carries a danger of long-delayed, fatal "slow virus" infection. The potential incidence of this is unknown. We

feel that the use of so called sicca cell therapy should be viewed as threatening to life.

The National Down Syndrome Congress provides this statement for your information and encourages the deepest of investigation prior to assumption of *any* therapy for a person with Down Syndrome in order to ascertain that therapy's efficacy and safety.

For further information or assistance, please call the National Down Syndrome Congress at 1-800-232-6372 or write to 1800 Dempster Street, Park Ridge, IL 60068-1146.

David A. Spence, M.D.
Juneau, Alaska

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AIR FORCE

President's Page

Greetings and Salutations and Happy New Year!

I had the privilege of attending the Interim Meeting of the American Medical Association in Atlanta, Georgia, along with Dr. Don Thieman, who is active in the Young Physician's section. The other members of ASMA present were Dr. Douglas Smith, our Delegate and Dr. Dave Johnson of Ketchikan, our Alternate Delegate. I must say we were very ably represented by these two physicians at the delegates meeting. Also attending was Ray Schalow, executive director of ASMA. He has formed some excellent liaisons between staff members of the AMA and our ASMA office. I feel that this will stand us in good stead in the years to come. Ray was quite active, attending the Reference Committee hearings and the House of Delegates sessions. Needless to say, the AMA House of Delegates looked at numerous problems facing the nation's physicians. Much time and effort was spent on the Harvard RVS (Relative Value Scale) study, and on developing a policy regarding AIDS treatment and testing of patients. Again, I came away very impressed with the quality of the organization that speaks for organized medicine in this country.

The Ad Hoc Convention Committee of ASMA/AAFP held its first meeting on December 15. Those attending were J.J. Smith, Peter Hansen, Robert Hall, myself, Ray Schalow, Ron Christensen, and Dale Trombley who is President of the AAFP organization. The group agreed to have a joint meeting in 1989 if we can work out details such as time, place, fiscal responsibilities and cost sharing. Because the meeting will be too large for Sitka, we discussed having the meeting in Juneau again. Juneau probably has the best facilities in the state for such a joint meeting. We look forward to working out the details for the project over the ensuring months. We will certainly discuss all aspects at the council meeting in Juneau in February.

I hope you have signed the initiative ballot for joint and several liability and that you have all been sponsors for this initiative. The end is at hand as far as getting more signatures.

Remember, the next annual meeting of ASMA will be in Fairbanks June 8-12, 1988.

I would like to congratulate John Middaugh, editor of *Alaska Medicine* and Verna Paluba, production editor, for the outstanding job they have done in improving the appearance and quality of our journal. The new editions are certainly eye catchers. We congratulate them and wish them well in their future endeavors.

Again, wishing you a Happy New Year.

Jerry Little, M.D.
President
Alaska State Medical Association

Executive Privilege

A significant event occurred during the fall Council meeting that may have gone unnoticed, but was a quantum leap in physician unity for Alaska. The Alaska State Medical Association Council unanimously approved a motion to admit United States Public Health Service physicians into association membership and, in addition, create a Councilor-at-Large seat. The House of Delegates, at its June meeting, will be asked to affirm this action.

By its action, the Council has flung open the doors of communication that have long been only ajar. It recognized that issues in the past have been communicated by innuendo or rumor. Now, through this action, ASMA has provided a forum to exchange ideas, agendas, and positions that need to be discussed.

Dr. Richard Mandsager, Director of the Alaska Native Medical Center, and Dr. C. Jerry Little, ASMA president, were principal forces in recognizing the need for interaction between physician groups. Dr. Mandsager initially was reluctant to accept an invitation to attend our 1987 Convention in Juneau. But, Dr. Mandsager was received as a peer and a colleague by our members which furthered his conviction of the benefits to USPHS physicians of membership in the Alaska State Medical Association.

Federal health services for Alaska Natives evolved after the departure of the Russians in 1867, when the Treaty of Cession was signed. This treaty insured that Alaska Natives would receive the same care as Indians elsewhere in the United States. But despite this provision, the Bureau of Indian Affairs did not take an active role in health matters until the 1930s. Instead, responsibility for health care was assumed by the federal government which could provide only fragmented care. But in the 1950's, all health services were transferred to the Bureau of Indian Affairs where they remain today. It is that long history of public service and medical experience that will add to the association's agenda for improving care for all the citizens of Alaska.

The Health Care Coalition of Alaska (HCC) continues to meet monthly to discuss issues of common interest. The six members of the Coalition include: Health Association of Alaska, Alaska Academy of Physicians Assistants, Alaska Public Health Association, Alaska Nurses Association, Alaska Pharmaceutical Association, and Alaska State Medical Association. This group of health service professionals represents the second largest employer group in the State of Alaska. It has made unified public statements to our legislators in Washington concerning the proposed Veterans Hospital or addition to Elmendorf AFB hospital. In the future the coalition will make public statements on AIDS and on sale of tobacco to minors, and it will attempt to create legislation to address these issues. HCC will address federal and state issues of substance, and actions will only occur with total unanimity.

One of the principal objectives of the HCC will be to encourage the Governor, legislature, and Department of Health and Human Services to create a long-range planning commission that will write a health policy for the State of Alaska.

A draft statement of purpose for HCC reads as follows:

> The Health Care Coalition of Alaska is committed to the betterment of the public health by developing, monitoring, and distributing information; by advocating health-related rights, responsibilities, and issues; and by representing our professions as a whole in an ethical and responsible manner which would bring honor to the health care profession. This is to be done in a manner that protects the autonomy of the individual members, improves the practice of health providers, and builds the public trust and confidence in the competence and reliability of our individual professions.

Raymond Schalow
Executive Director
Alaska State Medical Association

Auxiliary News

Alaska State Medical Association Auxiliary

It's that time of year again for the auxiliary to put on the Champ Camp raffle. As you recall, last year's raffle was a tremendous success. We were able to donate $7,620 to the Alaska Lung Association for this worthy camp. This year we hope to do as well.

In January and February, we will be collecting the terrific prizes from merchants. In March, ticket books will be provided to you for sale. Tickets will again be priced at $10 each in books of ten. I hope that every auxilian will be able to sell at least one book. If that happens, we should be able to almost double our donation.

Champ Camp is a wonderful community asset. Children with asthma meet for a week at Camp Peggy Lake. They learn to manage their asthma in a low key atmosphere while enjoying hiking, canoeing, boating, and other outdoor activities. This camp is a community effort, put on with volunteers, and we are fortunate to be able to participate.

By the time you read this, we'll know if the tort reform initiative qualified for the ballot. Thanks to all auxilians for your efforts in gathering signatures and sponsors.

Cassie Hill
President, ASMA Auxiliary

Letters to the Editor

Dear Editor:

I would like to comment on the letter in the November/December issue of *Alaska Medicine* by Elizabeth Fritz. I wholeheartedly agree that Alaska needs a medical museum. During a recent trip my husband and I visited a county medical museum in Massachusetts that was extremely interesting to both of us (both physician and non-physician) and is now listed as a tourist attraction although they do not charge a fee. It was apparently started by one retiring physician's donation of equipment, volunteer donation of time, and dollars from physicians and auxilians until today their collection covers all the walls and stairwells of two floors in their medical association building. I would like to see something similar developed here. Are there more of you "out there" also interested? Who could donate items, money, time and space to such an endeavor? Please send comments to the state medical office.

Gloria K. Park, M.D.
Anchorage, AK

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Editorial

A few weeks ago I spent another week (my fourth during the past two years) in Juneau examining death certificates. My time was spent primarily in reading each certificate and looking up the appropriate codes from the International Classification of Diseases (ICD) to assign to the cause of death and the other medical conditions listed on the death certificates. This time, I worked with four of my colleagues in the Section of Epidemiology, three entered the data into computers, one assisted in coding. We were able to code (and check, recheck, and recheck again) all death certificates for 1986, all out-of-state deaths 1980-86, and all deaths that occurred in Alaska in 1950. We also verified, corrected, and rechecked again the codes for all deaths from 1980-82 that we had coded last year.

All in all, we participated in an Iditadata of more than 10,000 death certificates. Tedious? Boring? You better believe it! Did we learn anything? We sure hope we will. But for now, although totally unscientific, let me share with you the impressions we took away along with conjunctival injection.

First, we have a splendid and dedicated group of professionals in our state vital statistics office who, working under difficult circumstances, have made major improvements in every aspect of collecting, recording, and processing vital records data. The foundation of all health policy and planning, our vital statistics office, is receiving long needed attention. Although we have a way to go, I believe we're on our way.

Second, there is a wealth of data just waiting to be turned into useful information that will assist us in meeting the challenges of delivering health services to Alaskans in the future.

Third, some things stand out.

- Alaska was a violent place in 1950. There were many homicides, drownings, and aircraft crash deaths.
- We have incredibly short memories—and some of us are young enough that our memories are pretty short. The number of deaths in 1950 from infectious diseases, especially tuberculosis, is mind-boggling. I found myself constantly searching the book for an array of infectious disease codes that I had not had to use for all the deaths I had coded for the years 1980-86. Sadly, I now know by heart the codes for rheumatic fever and heart disease, polio, measles, pertussis, abcesses, gangrene, tuberculosis and all its complications, diarrhea, peritonitis, and sepsis.
- Maternal death during childbirth was all too frequent and many newborn infants died with a diagnosis of "too small to live." Infant mortality was great not only from tuberculosis and measles but also from malnutrition, marasmus, failure to eat, and neglect, starvation, and the cold.
- Intracerebral hemorrhage and uncontrolled hypertension were all too frequently listed in 1950 as the cause of death. I can't remember coding any in 1986.
- Out-of-state deaths of Alaska residents in 1980-86 accounted for about 5-6% of all deaths each year. Many were newborn infants, many were elderly Alaskans with cancer. But, there were a surprising number of violent deaths—motor vehicle injury, suicide, and aircraft crashes.
- Including Alaskans who died flying to the lower 48 or while flying in the lower 48 states would raise our high aircraft fatality rates by a large proportion. Time for a hard look.
- In 1986 there were all too many fatalities at the worksite. Even a cursory look identified many deaths that should have been prevented. Time is long overdue for a hard look.

The Division of Public Health will be working on these data for many years to find clues for a healthier Alaska. But what progress we have made in the last 30 years!!! Our memories are short—too short. The improvement in the health of all Alaskans is nothing short of miraculous. We owe a tremendous debt to the health professionals who have contributed so much. But, there is no room for complacency.

Proper application of TODAY'S KNOWLEDGE can prevent two-thirds of potential years of life lost and can postpone two-thirds of all deaths. A look at the recent past can give us courage to continue our efforts to a healthier Alaska.

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Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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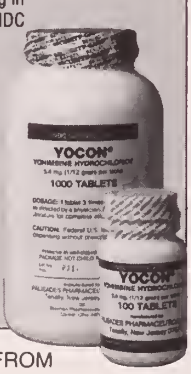

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There are no known contraindications to the use of sucralfate.

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Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients, adverse effects were reported in 121 (4.7%). Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

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References:
1. Grossman MI: *Scand J Gastroenterol* 58 (suppl 15):7-16, 1980.
2. Marks IN, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 70-81, 1984.
3. Krentz K, Jablonowski H, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 62-69, 1984.

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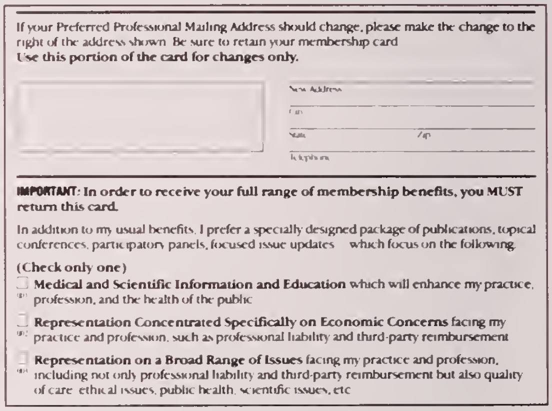

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Before prescribing, see complete prescribing information in SK&F LAB CO. literature or PDR. The following is a brief summary.

Contraindications: *There are no known contraindications to the use of 'Tagamet'.*

Precautions: *While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.*

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) Injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when 'Tagamet' is administered concomitantly. Interaction with phenytoin, lidocaine and theophylline has also been reported to produce adverse clinical effects.

However, a crossover study in healthy subjects receiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. h.s. concomitantly with a 300 mg. b.i.d. dosage of theophylline (Theo-Dur®, Key Pharmaceuticals, Inc.), demonstrated less alteration in steady-state theophylline peak serum levels with the 800 mg. h.s. regimen, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: *Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly unlikely. A single case of biopsy-proven periportal hepatic fibrosis in a patient receiving 'Tagamet' has been reported.*

How Supplied: Tablets: *200 mg. tablets in bottles of 100; 300 mg. tablets in bottles of 100 and Single Unit Packages of 100 (intended for institutional use only); 400 mg. tablets in bottles of 60 and Single Unit Packages of 100 (intended for institutional use only), and 800 mg. Tiltab® tablets in bottles of 30 and Single Unit Packages of 100 (intended for institutional use only).*

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Vials: *300 mg./2 ml. in single-dose vials, in packages of 10 and 30, and in 8 ml. multiple-dose vials, in packages of 10 and 25.*

Prefilled Syringes: *300 mg./2 ml. in single-dose prefilled disposable syringes.*

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ADD-Vantage®* Vials: *300 mg./2 ml. in single-dose ADD-Vantage® Vials, in packages of 25.*

Exposure of the premixed product to excessive heat should be avoided. It is recommended the product be stored at controlled room temperature. Brief exposure up to 40°C does not adversely affect the premixed product.

'Tagamet' HCl (brand of cimetidine hydrochloride) Injection premixed in single-dose plastic containers is manufactured for SK&F Lab Co. by Travenol Laboratories, Inc., Deerfield, IL 60015.

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Foods related to lowering the risk of cancer of the larynx and esophagus all have high amounts of carotene, a form of Vitamin A which is in cantaloupes, peaches, broccoli, spinach, all dark green leafy vegetables, sweet potatoes, carrots, pumpkin, winter squash and tomatoes, citrus fruits and brussels sprouts.

Foods that may help reduce the risk of gastrointestinal and respiratory tract cancer are cabbage, broccoli, brussels sprouts, kohlrabi, cauliflower.

Fruits, vegetables, and whole-grain cereals such as oatmeal, bra and wheat may help lower the ri of colorectal cancer.

Foods high in fats, salt- or nitrite-cured foods like ham, and fish and types of sausages smoked by trad tional methods should b eaten in moderation.

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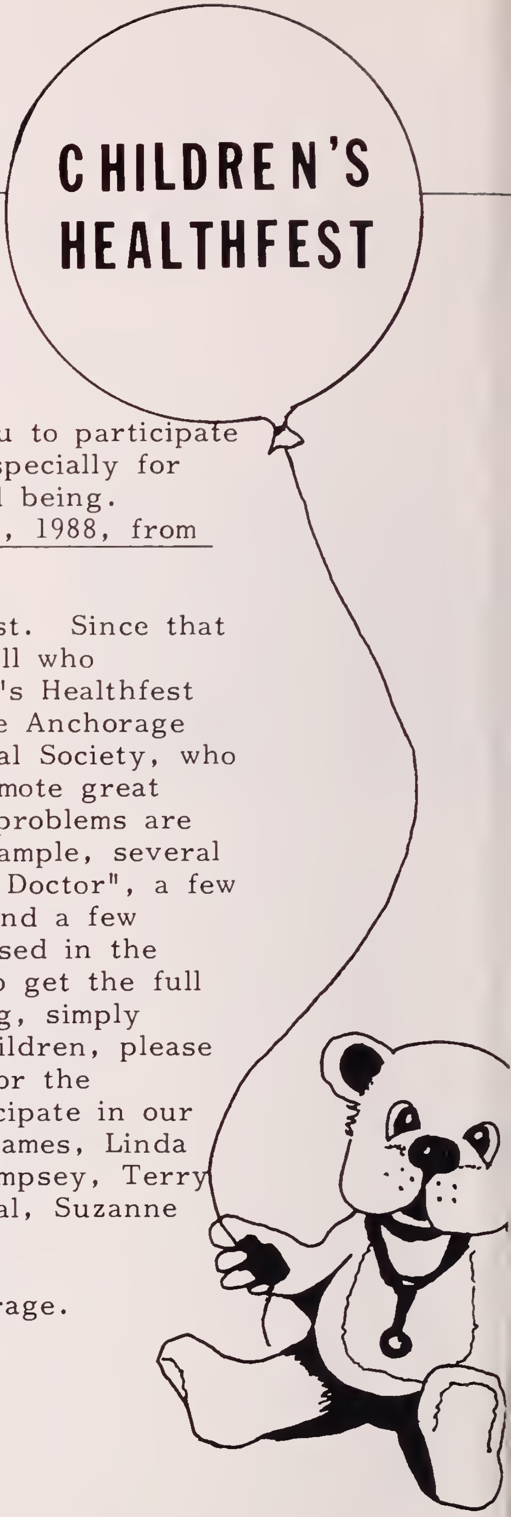

January 11, 1988

Dear Physician,

The Anchorage Medical Society and Auxiliary invite you to participate in our gift to the Anchorage area families, designed especially for children, to promote excellent health, fitness, and well being. Children's Healthfest will be held on Saturday, April 9, 1988, from 10 a.m. to 6 p.m., at the Northway Mall.

April, 1987 was our first year to sponsor the Healthfest. Since that time, only positive feed back has been received from all who participated, saying how wonderful a fair the Children's Healthfest really was. The fair consisted of participates from the Anchorage area, that had been approved by the Anchorage Medical Society, who demonstrated to children and their parents how to promote great health, fitness, and well being, and also, how health problems are treated if an accident or illness should occur. For example, several family physicians had a booth called "Ask your Family Doctor", a few orthopaedic surgeons demonstrated putting on casts, and a few surgeons demonstrated what kind of instruments are used in the operating room. (Complete with caps and face masks to get the full effect.) Before you count yourself out in participating, simply because your speciality does not "normally" involve children, please talk with one of us who is on the steering committee for the Healthfest. There is a way every physician can participate in our gift. We are; Tammy Horton, Cheri McGuire, Lynda James, Linda Sutherland, Alice Samuelson, Maria Baskous, Patty Dempsey, Terry McEvoy, Lindsay Tucker, Patrice Gerster, Annette Beal, Suzanne Boisen, and Jenna Hall.

We hope that you can participate in our gift to Anchorage.

Sincerely,

Tammy J. Horton

Tammy J. Horton
Chairperson, Children's Healthfest '88

Alaska Medicine, Volume 29, 1987

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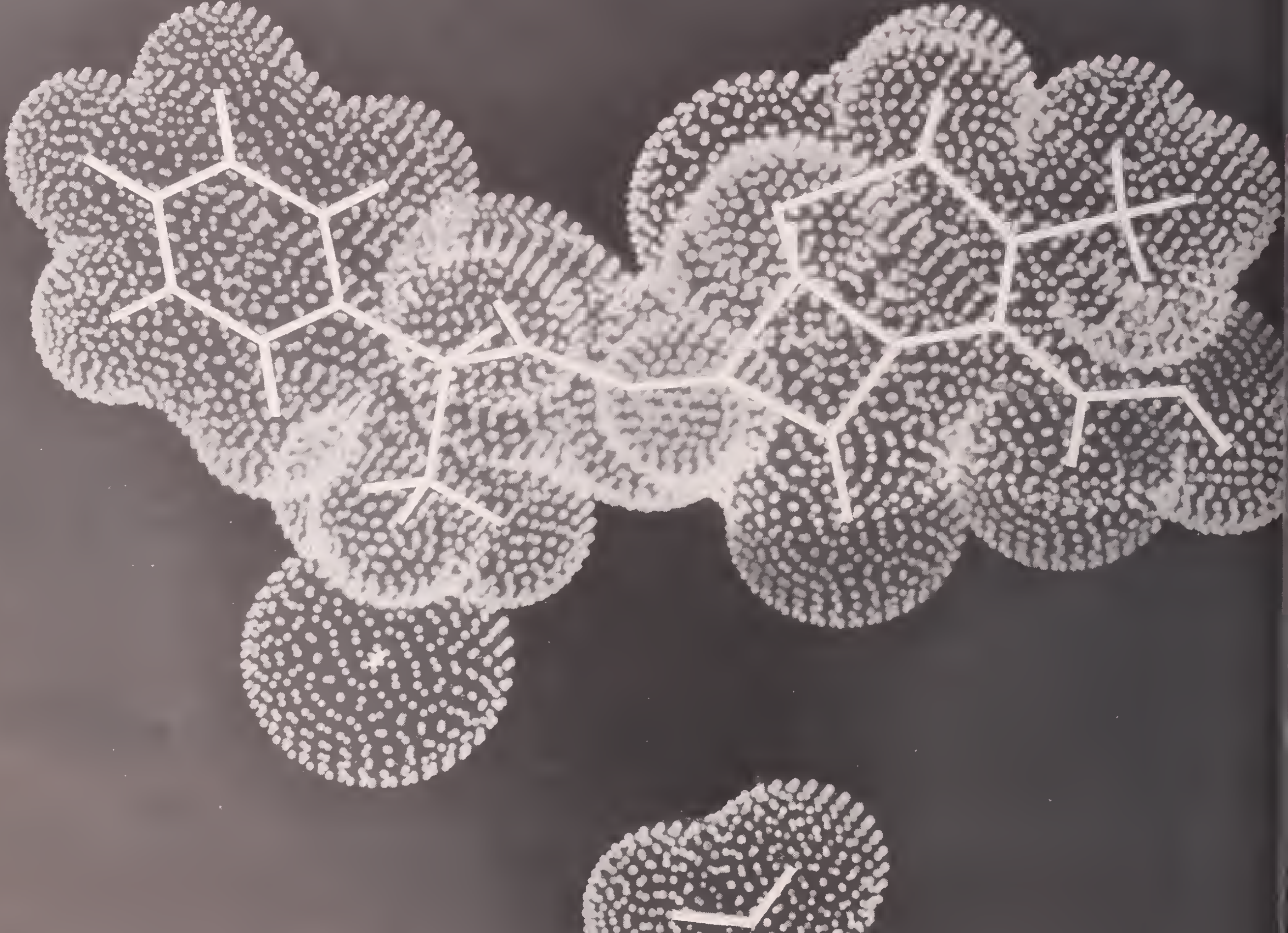
cephalexin hydrochloride monohydrate



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ncomplicated cystitis†
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ve patients.

lin is the drug of choice in the treatment and prevention
ptococcal infections, including the prophylaxis
ımatic fever.

susceptible strains of *Staphylococcus aureus* and/or β-hemolytic streptococci.
susceptible strains of *Escherichia coli*, *Proteus mirabilis*, and *Klebsiella* sp.
susceptible strains of group A β-hemolytic streptococci.

**KEFTAB™
(cephalexin hydrochloride monohydrate)**

Summary: Consult the package literature for prescribing information.

Indications and Usage:

Respiratory tract infections caused by susceptible strains of *Streptococcus pneumoniae* and group A β-hemolytic streptococci.

Skin and skin structure infections caused by susceptible strains of *Staphylococcus aureus* and/or β-hemolytic streptococci.

Bone infections caused by susceptible strains of *S aureus* and/or *Proteus mirabilis*.

Genitourinary tract infections, including acute prostatitis, caused by susceptible strains of *Escherichia coli*, *P mirabilis*, and *Klebsiella* sp.

Contraindication: Known allergy to cephalosporins.

Warnings: KEFTAB SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Keftab in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Keftab should be administered cautiously in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy and lactation. Cephalexin is excreted in mother's milk. Exercise caution in prescribing Keftab for these patients.
- Safety and effectiveness in children have not been established.

Adverse Reactions:

- *Gastrointestinal*, including diarrhea and, rarely, nausea and vomiting. Transient hepatitis and cholestatic jaundice have been reported rarely.
- *Hypersensitivity* in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens-Johnson syndrome, or toxic epidermal necrolysis.
- *Anaphylaxis* has been reported.
- *Other reactions* have included genital/anal pruritus, genital moniliasis, vaginitis/vaginal discharge, dizziness, fatigue, headache, eosinophilia, neutropenia, and thrombocytopenia; reversible interstitial nephritis has been reported rarely.
- Cephalosporins have been implicated in triggering seizures, particularly in patients with renal impairment.
- *Abnormalities in laboratory test results* included slight elevations in aspartate aminotransferase (AST, SGOT) and alanine aminotransferase (ALT, SGPT). False-positive reactions for glucose in the urine may occur with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (Glucose Enzymatic Test Strip, USP, Lilly).

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- **Significantly faster relief–62% of total four-week improvement evident in first week versus 44% with amitriptyline alone[1]**
- **Dramatic first-week reduction in somatic complaints[2]**

% Reduction in Somatic Symptoms[2]

Vomiting	Nausea	Headache	Anorexia	Constipation
Reduced 90%	Reduced 86%	Reduced 72%	Reduced 62%	Reduced 60%

- **Only ⅓ the dropout rate due to side effects of amitriptyline alone, although the incidence of side effects is similar[1]**

Caution patients about the combined effects of Limbitrol with alcohol or other CNS depressants and about activities requiring complete mental alertness, such as operating machinery or driving a car. In general, limit dosage to the lowest effective amount in elderly patients.

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References: **1.** Feighner JP, *et al* *Psychopharmacology 61* 217-225, Mar 22, 1979 **2.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ

Limbitrol® Ⓒ IV
Tranquilizer—Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs Closely supervise cardiovascular patients (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses Myocardial infarction and stroke reported with use of this class of drugs.) Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e g , operating machinery, driving).

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage, withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide)

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients Periodic liver function tests and blood counts are recommended during prolonged treatment Amitriptyline component may block action of guanethidine or similar antihypertensives When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady state concentrations of the tricyclic drugs Concomitant use of Limbitrol with other psychotropic drugs has not been evaluated, sedative effects may be additive Discontinue several days before surgery Limit concomitant administration of ECT to essential treatment See Warnings for precautions about pregnancy Limbitrol should not be taken during the nursing period Not recommended in children under 12 In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects

Adverse Reactions: Most frequently reported are those associated with either component alone drowsiness, dry mouth, constipation, blurred vision dizziness and bloating Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely

The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs

Cardiovascular Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke

Psychiatric Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns

Anticholinergic Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract

Allergic. Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus

Hematologic Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia

Gastrointestinal Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion

Other Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling

Overdosage: Immediately hospitalize patient suspected of having taken an overdose Treatment is symptomatic and supportive I V administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning See complete product information for manifestation and treatment

Dosage: Individualize according to symptom severity and patient response Reduce to smallest effective dosage when satisfactory response is obtained Larger portion of daily dose may be taken at bedtime Single *h.s* dose may suffice for some patients Lower dosages are recommended for the elderly Limbitrol DS (double strength) Tablets, initial dosage of three or four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required Limbitrol Tablets, initial dosage of three or four tablets daily in divided doses, for patients who do not tolerate higher doses

How Supplied: *Double strength (DS) Tablets*, white, film-coated, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt), and *Tablets*, blue, film-coated, each containing 5 mg chlordiazepoxide and 12 5 mg amitriptyline (as the hydrochloride salt) Available in bottles of 100 and 500, Tel-E-Dose® packages of 100, Prescription Paks of 50

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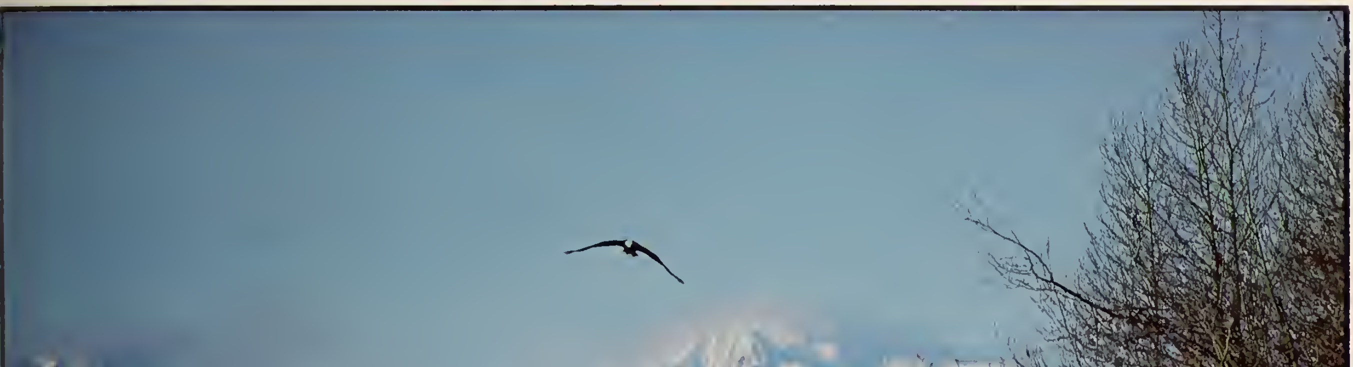

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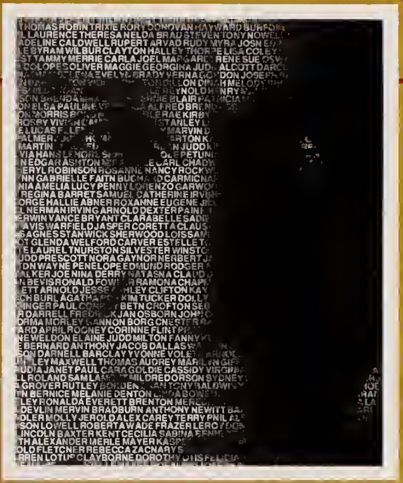

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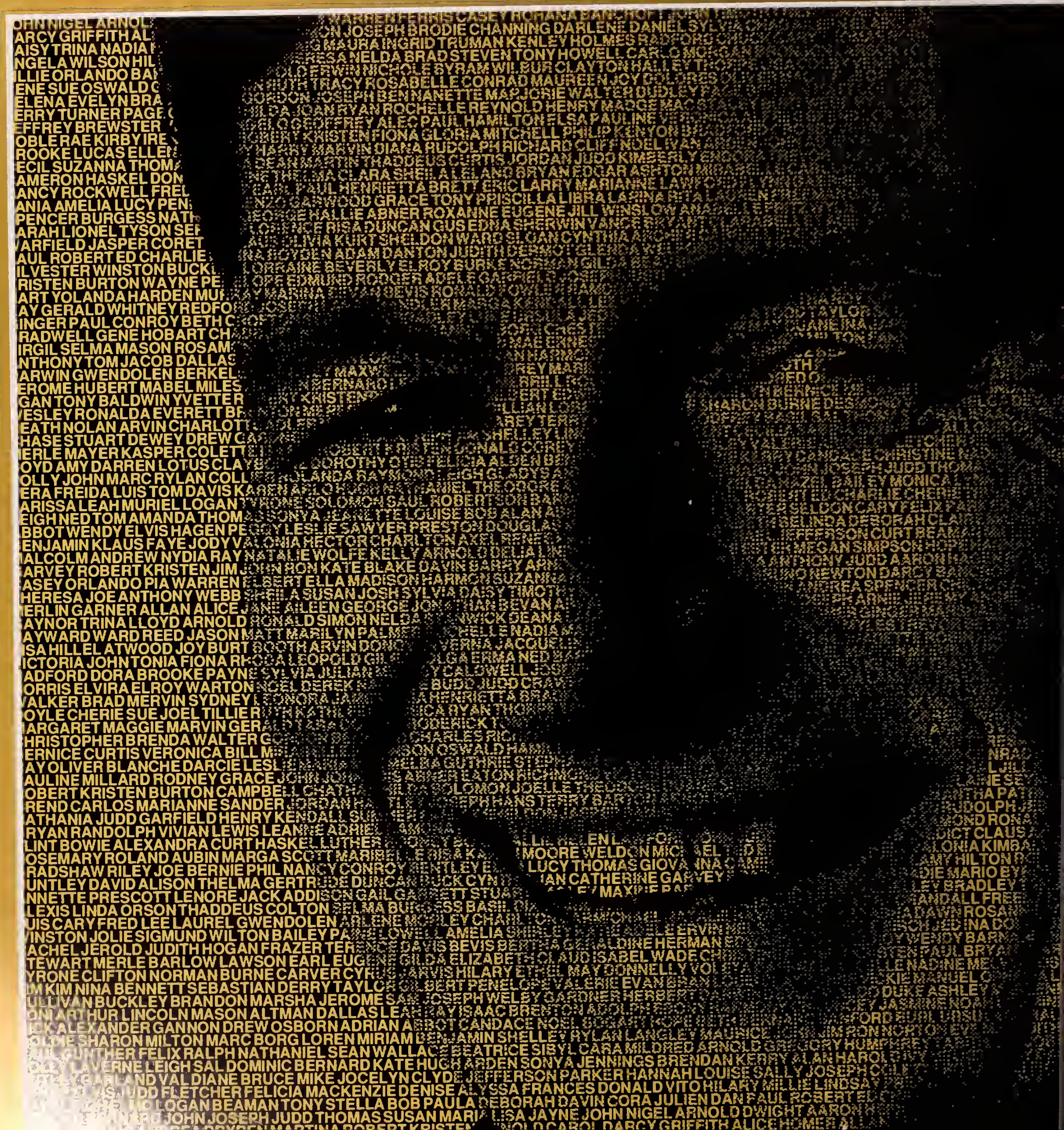
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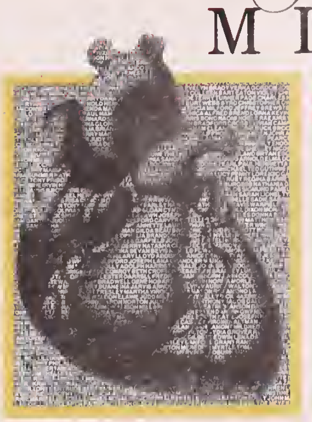

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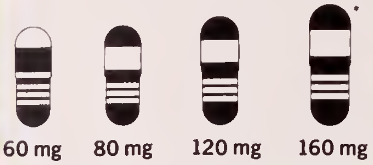

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CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) tor the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose ot INDERAL Tablets. The lower AUCs tor the capsules are due to greater hepatic metabolism ot propranolol, resulting trom the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute tor conventional propranolol and the blood levels achieved do not match (are lower than) those ot two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need tor retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure, and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management ot hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated for the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is useful in the management ot hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness ot propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first-degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory tunction in patients with congestive heart tailure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart tailure, it necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following ab*rupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management ot unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to tollow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)—PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY: The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery ot blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs of hyperthyroidism. Therefo
abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyro
ism, including thyroid storm. Propranolol may change thyroid function tests, increasing T_4
reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reportec
which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a dema
pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impai
hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypert
sive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be t
that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return
increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disea
elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as res
pine should be closely observed if INDERAL (propranolol HCl) is administered. The add
catecholamine-blocking action may produce an excessive reduction of resting sympathe
nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attac
or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calciu
channel-blocking drug, especially intravenous verapamil, tor both agents may depress myocard
contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use
beta blocker and verapamil has resulted in serious adverse reactions, especially in patients
severe cardiomyopathy, congestive heart failure, or recent myocardial infarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate ot absorption of propranolol.

Phenytoin, phenobarbitone, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma level
both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly
propranolol.

Cimetidine decreases the hepatic metabolism of propranolol, delaying elimination and increas
blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in anim
have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in b
rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug
duced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reprod
tive studies in animals did not show any impairment ot fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in ani
studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be us
during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised w
INDERAL is administered to a nursing woman.

PEDIATRIC USE: Satety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have ra
required the withdrawal of therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensification of AV block; hypotensi
paresthesia of hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud ty

Central Nervous System: Light-headedness; mental depression manifested by insomnia, lassitu
weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; ha
cinations; vivid dreams; an acute reversible syndrome characterized by disorientation for time
place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased per
mance on neuropsychometrics. For immediate formulations, fatigue, lethargy, and vivid drea
appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, consti
tion, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching
sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence,
Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the s
serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associa
with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride
sustained-release capsule for administration once daily. If patients are switched from INDEF
Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effe
maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDER
INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necess
especially to maintain effectiveness at the end ot the 24-hour dosing interval.

HYPERTENSION—*Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL
once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg o
daily or higher until adequate blood pressure control is achieved. The usual maintenance dosag
120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needec
full hypertensive response to a given dosage is variable and may range from a few days to sev
weeks.

ANGINA PECTORIS—*Dosage must be individualized.* Starting with 80 mg INDERAL LA once d
dosage should be gradually increased at three- to seven-day intervals until optimal respons
obtained. Although individual patients may respond at any dosage level, the average optimal dos
appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320
per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (
WARNINGS).

MIGRAINE—*Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once d
The usual effective dose range is 160-240 mg once daily. The dosage may be increased graduall
achieve optimal migraine prophylaxis. If a satistactory response is not obtained within four tc
weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It ma
advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS—80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE—At this time the data on the use of the drug in this age group are too limite
permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

Reference:
1. Data on file, Ayerst Laboratories.

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Circumpolar Health – 87

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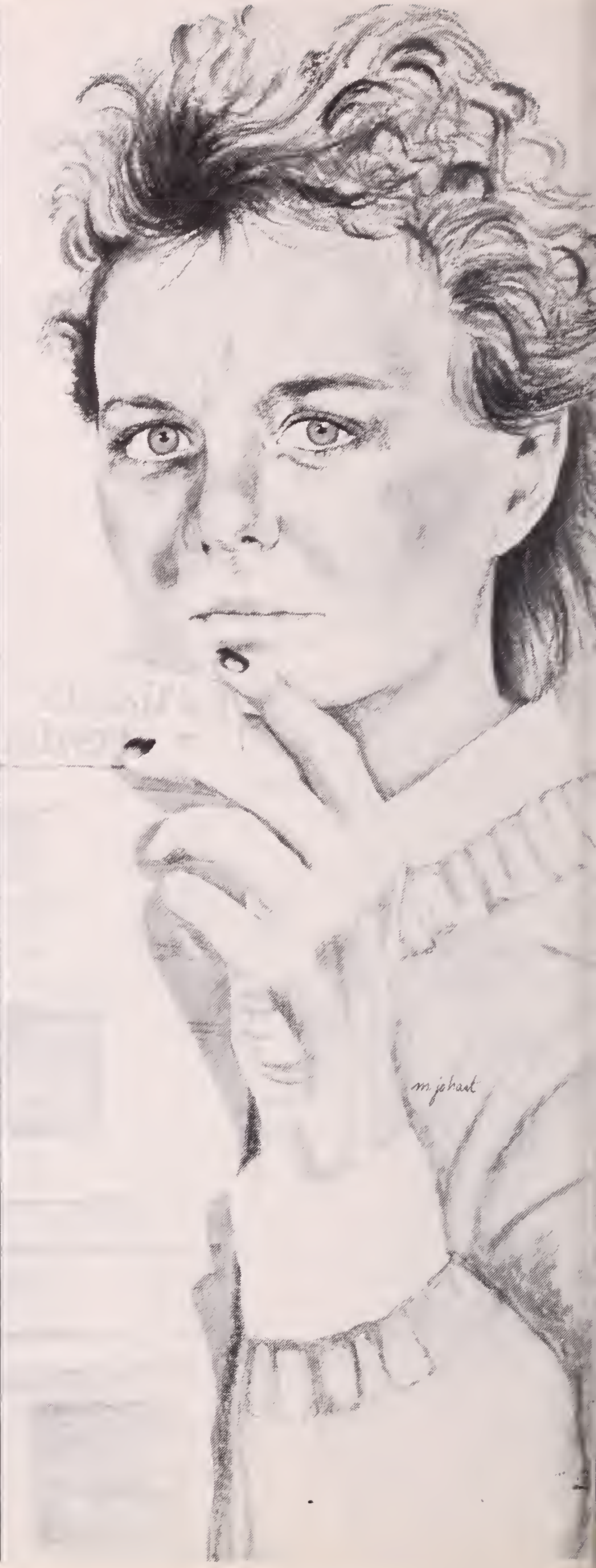

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An Outbreak of Trichinosis Traced to Alaskan Black Bear Meat

by Thad L. Woodard, M.D.[1]

ABSTRACT

Investigation of a case of trichinosis subsequently led to the discovery of 21 other cases. These were among 65 individuals attending a total of seven meals at which meat from a single Alaskan black bear was served in two states over a 75-day period. Meat from this bear was found to have an extremely high *Trichinella spiralis* larval load of 1200 larvae per gram of tissue. Attack rates based on method of meat preparation ranged from 0 percent to 88 percent (overall attack rate 65 percent). The high larval load and inadequate preparation were critical factors in the high attack rates as well as the high percentage (82 percent) of symptomatic individuals. Further evidence of the cold resistance of arctic trichina is presented.

INTRODUCTION

That trichinosis can result from the ingestion of improperly prepared pork or pork products is well known; however, many individuals fail to remember that *Trichinella spiralis* is a parasite of many species of animals, particularly carnivorous mammals (1). For this reason, not only common meats but also meats prepared from wild or exotic animals may provide substantial risk of acquiring trichinosis (2–4), especially if these meats are served raw or slightly cooked. Furthermore, because such exotic meats are frequently served to large gatherings of adventuresome people, outbreaks may be explosive and extend widely over time and place. The outbreak described in this report illustrates these principles.

Description of the Outbreak

Figure 1 shows the time sequence of important events in the outbreak. On September 24, 1978, a 400-pound, male black bear was killed near Tyonek, Alaska, a village 40 miles west and across Cook Inlet from Anchorage, and meat from this bear was given to three individuals in Anchorage. In each instance the meat was kept in freezer units at approximately 0° F (–18° C) until just before use in a meal. The first meal at which the bear meat was served occurred on October 1, one week after the kill. Subsequent meals in Anchorage where the meat was served occurred on October 14, October 19, November 4, November 11, and December 9. (Numbers in parentheses in Figure 1 indicate the number of people attending each meal).

The index case became ill and was diagnosed as having trichinosis at the Los Angeles County—University of Southern California General Hospital on November 19, eight days following the fifth meal. The case was reported to the Los Angeles County Health Department, and because the patient had eaten bear meat in Anchorage at meals 2, 3, and 5, the Alaska Division of Public Health was notified on December 6.

The family in Anchorage who had prepared these meals was contacted; however, the family representative denied knowledge of other symptomatic individuals and furthermore stated that since she was an Alaskan Native and aware of the risk of acquiring trichinosis from bear meat, the meat had been thoroughly cooked. Because of this finding it was suggested to the investigator in Los Angeles that the patient may have had another exposure.

However, 27 days later, on January 2, 1979, a physician at the Alaska Native Medical Center in Anchorage reported to the Alaska Division of Public Health another person with trichinosis who also had eaten the bear meat. An interview with this patient established that he had eaten bear meat at meal 5, one of the meals the index case had also incriminated. The family who prepared this meal was again contacted and admitted that several family members currently had symptoms of trichinosis.

It was further learned that a portion of the meat had also been taken to Los Angeles in December so the Los Angeles Health Department was renotified. In Los Angeles it was discovered that this meat was served at a seventh meal on December 23. Thus, by the time the full investigation had begun and all remaining meat had been confiscated, the bear meat had been served at seven meals attended by 65 people in two states over a period of two and one-half months.

Methods and Materials

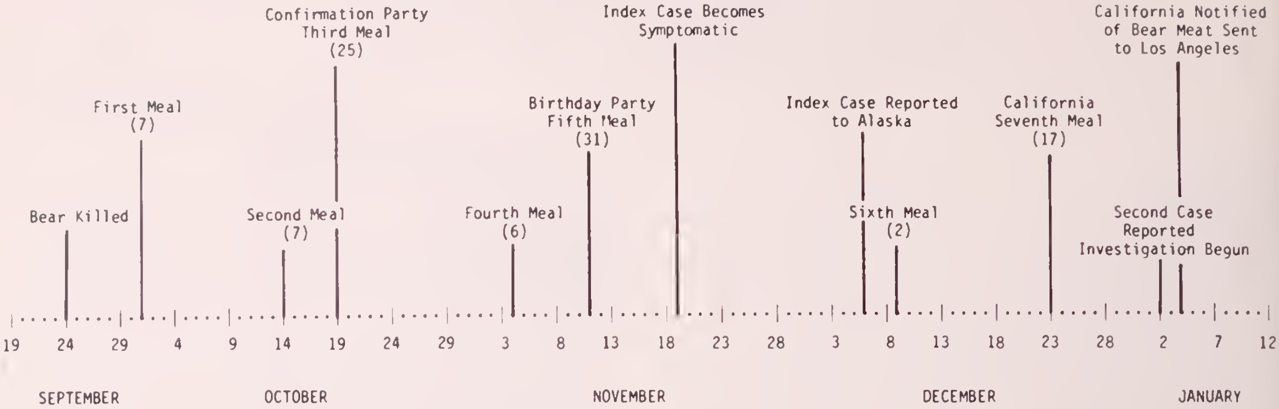
Epidemiologic investigation. Individuals involved in the preparation of each of the meals were contacted and questioned about the menu served and the names of individuals attending each meal. Since the bear meat was the only likely infected food served, detailed information regarding the storage and preparation of this food was obtained.

An attempt was made to contact each of the 65 individuals known to have attended the meals and to admin-

---

[1] From the Field Services Division, Bureau of Epidemiology, Centers for Disease Control and Epidemiology Office, Alaska Division of Public Health. Dr. Woodard is now in the private practice of pediatrics, 1200 Airport Heights, Suite 140, Anchorage, Alaska 99508.

Figure 1. Chronology of Trichinosis Outbreak – Alaska, California



ister a questionnaire designed to elicit a history of signs and symptoms of trichinosis as well as the date of onset. The interviewers also asked whether the individual had eaten bear meal at any of the suspected meals and if so, how the meat was prepared. If possible, blood was obtained for a complete blood count and differential as well as trichinosis bentonite flocculation test.

Laboratory investigations. Trichinosis bentonite flocculation testing was done on serum by the Parasitic Serology Branch, Parasitology Division, Bureau of Laboratories, Center for Disease Control (5). Complete blood counts with differential were done on EDTA—anticoagulated whole blood.

No meat prepared at any meal was available for examination; however, meat from the same bear remained frozen in Anchorage and was confiscated. A 10-gram portion of this meat was digested in trypsin hydrochloride and examined for trichina cysts (6). This testing was provided by the U.S. Department of Agriculture Veterinary Services, Palmer, Alaska, and confirmed by the Center for Disease Control.

Case Definition. Individuals were considered cases if they gave a history of eating the implicated bear meat at any of the suspect meals and had either clinically compatible symptomatology or any of the following laboratory findings: a fourfold trichinosis bentonite flocculation titer change; a single diagnostic bentonite flocculation titer $\geq$1:5; or eosinophilia $>$5 percent.

RESULTS

Epidemiologic investigations. The location, date, and method of bear meat preparation for each meal is summarized in Table 1. The first six meals occurred in Anchorage and the last in Los Angeles. For the first meal a single 2–3-pound portion of meat was boiled for 60–90 minutes. In the remaining meals in Anchorage small slices of meat were quick-fried or seared using a Korean utensil called a "buy koki pan" (Figure 2). This utensil is placed over an electric heat source and marinated meat slices are then placed on the heated convex surface. Interviews established that the majority of meat prepared in this method was simply seared on both sides prior to ingestion. However, at meal 3 a 1–2-pound roast of bear meat baked at 350° for 3 hours was also present, and at meal 6 the meat was cooked more slowly for 30–45 minutes, on the "buy koki pan." At meal 7 in Los Angeles small "beef stew" size chunks of meat were cooked up to 10 minutes on a charcoal grill.

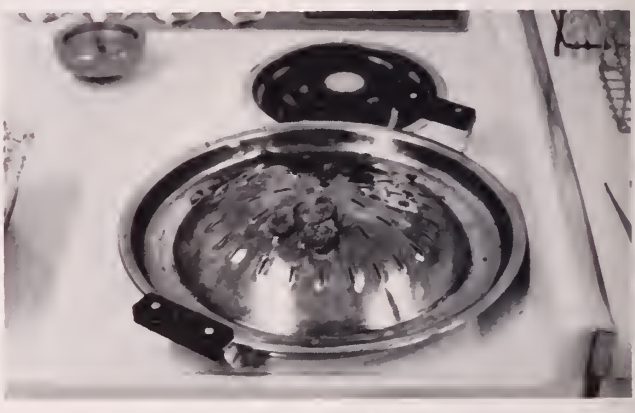


Figure 2. "buy koki pan"

Of the 65 people who attended the seven meals, 63 were contacted and interviewed; 54 had blood samples obtained for complete blood count and differential plus bentonite flocculation testing. Thirty-four of the 65 recalled eating bear meat at the meals. Of these 34 considered at risk, 22 had symptoms that fit the case definition, giving an attack rate of 64.7 percent for those who ate the implicated meat.

Cases occurred in individuals eating only at meals 2,3,5, or 7 and having no other known exposure, clearly suggesting infection occurred at multiple meals and perhaps at all seven meals. However, calculating attack rates for each of the various meals was complicated by the fact that many of the persons with trichinosis had eaten at multiple meals. Therefore attack rates based upon the method of bear meat preparation were calcu-

Table 1. Method of bear meat preparation at each meal

Meal	Location	Date	Preparation
1	Anchorage	October 1	Boiled 60-90 minutes
2*	Anchorage	October 14	Buy koki pan up to 10 minutes.
3*	Anchorage	October 19	Buy koki pan up to 10 minutes/ Baked 350°F 3 hours
4	Anchorage	November 4	Buy koki pan up to 10 minutes
5*	Anchorage	November 11	Buy koki pan pan up to 10 minutes
6	Anchorage	December 5	Buy koki pan 30-45 minutes
7*	Los Angeles	December 23	Charcoal grilled up to 10 minutes

***Indicates meal resulting in a case of trichinosis having no other known exposure.**

lated (Table 2). In this way cases among individuals who ate bear meat at multiple meals could be examined.

Seven cases and 2 non-cases were in persons eating meals at which the bear meat was prepared using the "buy koki pan" only, giving an attack rate of 78 percent for this preparation. The attack rate was 67 percent among people eating meals for which the meat was prepared on the "buy koki pan" or was baked or boiled. There were no cases among individuals eating only boiled bear meat. The attack rate at the meal in California where only charcoal grilled meat was served was 88 percent.

These data strongly suggest that meat prepared in Anchorage in the "buy koki pan" and charcoal grilled in Los Angeles was still infectious prior to ingestion. Only for the first meal in Anchorage, where only boiled meat was served, was the preparation adequate. No ill person ate only baked meat, but this preparation should have been more protective. The attack rate disregarding those eating only boiled meat was 76 percent.

Of the 22 total cases, 13 were Alaskan Natives, and 9 were Korean; their ages ranged from 7 to 61 years. No significant sex difference was present between cases and individuals at risk.

Table 2. Attack rates based upon method of bear meat preparation

Preparation	Case	Non-case	Attack Rate
Buy koki pan only	7	2	78%
Buy koki pan/baked/broiled	8	4	67%
Boiled only	0	5	0
Charcoal grilled only	7	1	88%

To determine the incubation period of the illness in this outbreak, only the California cases were used since there were only 4 symptomatic persons in Alaska for whom the exact date of exposure could be determined. In the seven cases considered, the incubation period ranged from 7 to 28 days (mean 17 days).

The most common complaints among symptomatic persons were fever/chills and myalgias, each occurring in approximately 70 percent of the ill persons (Table 3). Periorbital edema and diarrhea occurred in 45 percent of symptomatic patients. Gastrointestinal complaints were a common symptom. Only 4 of 22 cases (18 percent) were asymptomatic. Three patients were hospitalized, two in Alaska and one in Los Angeles. All eight ill persons in California and two in Alaska received thiabendazole.

Table 3. Symptom frequency among 22 cases of trichinosis

	Patients	Percent
Fever/Chills	16	73
Myalgia	15	68
Weakness/Easy Fatigue	13	59
Periorbital Edema	10	45
Diarrhea	10	45
Abdominal Cramps	8	36
Nausea/Vomiting	7	32
Rash/Urticaria/Pruritus	7	32
Visual Disturbance	5	23
Sore Throat/Mouth	3	14

Laboratory investigations. Eosinophilia occurred in 77 percent of the patients and ranged from 6 percent to 64 percent (mean 32 percent). Either a single diagnostic bentonite flocculation titer or a significant change in titers occurred for 70 percent of cases. Only 2 of 32 (6.2 percent) individuals bled and not considered cases had eosinophilia, and none of these had serologic evidence of infection.

Table 4 summarizes the eosinophilia and bentonite flocculation results for 20 of the 22 cases. For 19 of 20 cases both tests were in agreement. In one instance serologic evidence of infection was absent 48 days after the last known exposure in an individual with 64 percent eosinophilia. In five patients considered cases on the

Table 4. Bentonite flocculation results and eosinophilia in 20 cases of trichinosis

	Bentonite flocculation (titer $\geq$1:5)		
Eosinophilia (> 5%)	Positive	Negative	Total
Positive	14	1	15
Negative	0	5	5
Total	14	6	20

basis of symptomatology, both tests were nondiagnostic.

Trypsin hydrochloride digestion of the bear meat revealed 1220 cysts per gram, an extremely high larval load. It is quite rare to find more than 300–400 cysts per gram of bear meat in Alaska.

DISCUSSION

Human trichinosis following consumption of bear meat has long been a problem in the arctic and Alaska. Even in recent times, outbreaks and cases of bear-meat-related trichinosis continue to occur in Alaska (2,7). In fact, while evidence accumulates that incidence and prevalence of human trichinosis is declining in the continental U.S. (8), this outbreak and other recent outbreaks associated with walrus (4) indicate at least a constant and perhaps growing problem exists in Alaska. Furthermore, we know of no evidence to suggest the high prevalence of trichinosis in bears in Alaska has declined since prior observations, and recent walrus-associated outbreaks are leading to a reassessment of the prevalence of trichinosis in this animal.

Outbreaks associated with bear meat and other exotic meats often involve rather large numbers of people. The ease of modern transportation has made possible wide dissemination not only of infected individuals but also infected meat as this outbreak demonstrates. This dissemination also complicates appropriate public health action.

In this outbreak the extremely high larval load of the infected meat placed added importance on appropriate preparation of the meat. Unfortunately, preparation in most instances was not adequate. This inadequate preparation combined with the high trichina load presumably was critical in increasing the infectious dose causing not only a high overall attack rate but also a high rate (82 percent) of symptomatic infection.

USDA regulations indicate *T. spiralis* in pork held at 5° F (–15° C) for 20 days is rendered nonviable (9). In this and other outbreaks, epidemiologic investigations have suggested that *T. spiralis* found in arctic mammals remains infectious after storage at temperatures and times that exceed USDA requirements (2,4). Infected bear meat from this outbreak was forwarded to the Parasitic Disease Division, Bureau of Epidemiology, CDC, to be used in studies to test the hypotheses that the arctic strain of *T. spiralis* is cold-resistant. Portions of this bear meat held at 5° F (–15° C) for up to 35 days contained larvae still as infectious for laboratory animals as samples from the same meat refrigerated at 10° C for the same lengths of time (10). It is not known whether this apparent resistance to cold is related to the physical differences between bear meat and pork or a separate characteristic of this strain.

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Accidental Deaths and Suicides in Southwest Alaska: Actual versus Official Numbers

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ABSTRACT

This article reports the results of a study of accidental deaths and suicides over the period 1979-84 in 49 predominantly Native villages in southwest Alaska. It shows the discrepancies between the actual and official numbers resulting from misrecording, underrecording, and underreporting, and explains how they happened. The characteristics of the population most at risk for suicide are described.

1.0 INTRODUCTION

The authors recently completed a study (1) for the Yukon-Kuskokwim Health Corporation (YKHC) which provides health care for the residents —predominantly Yup'ik-speaking Eskimos— of 49 villages in southwest Alaska.

The YKHC notes that "...suicide (in its region) is not well documented and statistics do not accurately reflect the actual situation (2)." The Alaska Department of Health and Social Services (DHSS) notes that, under certain assumptions, "...the official records would be underestimating suicides by approximately 80% (3)."

The YKHC wanted to: find out if the actual numbers differed from the official numbers, uncover whatever patterns there might be in the epidemiology and etiology of suicide in its region, institute procedures that would yield more reliable numbers, and see how its region compared with others. The authors' study addressed those topics.

Based on it, this article explains how and why the official and actual numbers of accidental death and suicides differ, describes the characteristics of the regional population most at risk for suicide, and compares Native rates in the region with Native rates elsewhere.

2.0 METHODS

A six-year period was accepted by the YKHC as adequate for purposes of revealing discrepancies; 1979-84 was chosen because 1984 was then the latest year for which official numbers were available.

Two sources of data were compared: DHSS printout of violent deaths (4), and registers of death and coroners' files in the villages.

The DHSS printout, requested by the authors, listed all violent deaths by name, village of residence, sex, and race of decedent. It included five categories of death; accident, homicide, suicide, other, and undetermined motive. For each decedent it gave the three- or four-digit, ICD-9, cause-of-death code (5) with brief accompanying description (for example: 910.8, water transport-drown).

The registers of death contained annual lists of all deaths and certificates of death. The coroners' files included: certificates of death, certificates of fetal death, certificates of presumptive death, autopsy reports of pathologists, inquest proceedings, orders to dispense with inquests, affidavits for correction of a record, reports of their investigations by State troopers and city police, and correspondence by the decedents.

3.0 RESULTS

Table 1 shows the discrepancies between the printout (referred to as VS for Vital Statistics) and the information found in the files (referred to as CF for Coroners' Files).

VS (line 1) had 220 individuals resident in the region who died in the region as a result of an accidental death

Table 1. Accidental deaths and suicides, vital statistics (VS) versus coroners' files (CF)

	accidental deaths			suicides		total
1. vital statistics (VS)			215		5	220
2. minus			51		0	51
3. misrecording		40				
4 suicides	24					
5. homicides	16					
6. overrecording		3				
7. double-counting	2					
8. non-human remains	1					
9 overreporting		8				
suicides	8					
10. equals			164		5	169
11. plus			37		33	70
12. misrecording				24		
13. underrecording		37		1		
14. underreporting				8		
15. equals coroners' files (CF)			201		38	239

Sources: VS - Master Tape Violent Deaths for the Calista Region sorted by year, town of residence. November 15, 1985. Alaska Department of Health and Social Services. Juneau, AK.

CF - Coroners' Files

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or a suicide: 215 who died as a result of an accident; five who committed suicide.

CF (line 10) shows that, of the 215 accidental deaths recorded by the VS, only 164 were accidental deaths. The 51 deducted consist of 40 that were misrecorded (line 3), three that were overrecorded (line 6), and eight that were overreported (line 9). The 40 misrecorded consist of 24 locally-determined suicides and 16 locally-determined homicides. The three overrecorded consist of two that were counted twice, and one classified by the pathologist as non-human remains. The eight overreported were all deaths that the authors determined to be suicides despite the fact that the files on these deaths showed that they had been determined locally to be accidental deaths. (These eight, and the reasons for the authors' determinations, are discussed in section 5.1). These 51 (40 misrecorded, three overrecorded, and eight overreported) were deleted for a subtotal of 164 accidental deaths. To them were added 37 accidental deaths recorded as such in the coroners' files but not included in the DHSS printout on violent deaths, for a total of 201 actual accidental deaths (line 15).

The VS and the CF were in agreement on five suicides. The 33 additions consist of: the 24 misrecorded as accidental deaths; one death found in the files (which the authors determined to be a suicide, see section 4.2.2) but not in the DHSS printout; the eight overreported accidental deaths.

In sum, VS and CF agreed on 164 accidental deaths out of a total of 201, and on five suicides out of a total of 38.

4.0 ANALYSIS

These results focus the analysis on misrecording and underrecording. (Overrecording, with three cases, is minor.)

4.1 Misrecording

Here the question is: Why did the DHSS printout of violent deaths misrecord 24 suicides and 16 homicides (Table 1, lines 4 and 5) as accidental deaths?

There are three reasons. One is that the National Center for Health Statistics coded incorrectly because of inadequate information on the certificate of death. The second is that there was poor communication between the local coroners and DHSS. The third is that DHSS mishandled the data. These three are discussed each in turn in this section.

4.1.1 INCORRECT CODING BY THE NCHS

One example—of a suicide—is typical of the 24 suicides and 16 homicides misrecorded as accidental deaths. This suicide is misrecorded on the DHSS printout which shows it as an accident: 9229, accident firearms.

The printout is compiled from ICD-9, cause-of-death codes selected by NCHS nosologists, entered by them on certificates that NCHS returns to DHSS. (Alaska is one of 35 states that use this procedure; the other states do their own coding according to specifications established by NCHS, which also undertakes quality control (6). Since November, 1985 DHSS has employed a nosologist-trainee who may both query the coroners and code the certificates before their transmittal to NCHS.)

In selecting the code for the cause-of-death, NCHS establishes also the manner-of-death. Its three- or four-digit cause-of-death codes belong to one or the other of six major groups of manner-of-death that constitute the universe of possibilities: 0-799, natural; E800-949, accidental; E950-959, suicide; E960-978, homicide and legal intervention; E980-989, undetermined; E990-999, injury from war.

On the 1979 certificate of death, which is all NCHS had, two causes of death are given: internal extravasation; gunshot wound abdomen. The incident could have been an accident, a homicide, or a suicide. In 1981 a local inquest determined it to be a suicide. The 1985 DHSS printout, based on the code selected by NCHS in 1981, still shows this as 9229: accident firearms.

This early certificate did not have any of the four boxes for manner-of-death filled in locally: accident, homicide, suicide, or undetermined. Certificates that do have such boxes filled in are the exception rather than the rule. (This is discussed in section 4.2.1 as a critically-important omission.) The accident code was therefore NCHS' best guess.

However, there was other information available at the time —not included on the certificate— that might have led to a correct code if it had been included. The autopsy report, which the coroner receives before he sends the certificate to DHSS (who send it on to NCHS), gave two different causes of death: gunshot wound, head, consistent with self-inflicted; gasoline in gastric content. If these causes instead had been on the certificate, the agencies may have queried the local coroner and NCHS may then have selected the correct code: suicide.

In any case, this example makes the point that NCHS coded incorrectly because the certificate of death contained inadequate information. With homicides, it has the result that individuals are found guilty and incarcerated for incidents that still appear in the vital statistics as accidents.

4.1.2 POOR COMMUNICATION

The above example indicates that the coroner did an inadequate job of communicating the causes of death. This poor communication was compounded later. The coroner's file on this case contains no Affidavit for Correction of a Record (i.e. the early certificate) that should have been prepared by the local coroner after the inquest and then sent to DHSS. The authors found numerous such omissions. Symptomatic of this is that, in one series of files, a judge had taken it upon himself to attempt to bring files up to date by issuing Orders to Dispense with Inquest —years after the incidents— making at that time the first local determination of manner-of-death: as an accident. Finally, until November, 1985, when it hired a nosologist-trainee, DHSS appears to have had no procedure in place for communicating with local coroners: querying certificates sent by coroners that were likely to cause coding difficulties when forwarded to NCHS.

4.1.3 DHSS PROBLEMS

Even with good communication, an initial error in coding may remain in the vital statistics because of data-handling problems within DHSS. "Information which came in after the yearly tape was created was not routinely added [by way of correction and, because] the master tape...would not accept changes in the cause-of-death code...updated information...did not appear in the computerized files (7)." That is, even when coroners sent affidavits (or other forms of correction) the corrections to the vital statistics may not have been made.

4.2 Underrecording

The word is used here to refer to the 37 accidental deaths and the one suicide (Table 1, line 13) that were not recorded in the vital statistics as violent deaths.

Here, the question is: Why are these 38 incidents missing from the DHSS printout on violent deaths?

There are two main reasons. One is that NCHS coded many, incorrectly, as natural deaths. The other is that problems within DHSS resulted in the exclusion of some NCHS coded, correctly, as accidental deaths. These facts were established by examination of NCHS-coded certificates, copies of which the authors obtained from DHSS.

4.2.1 INCORRECT CODING BY THE NCHS

Of the 37 accidental deaths, the authors examined coded certificates for 34. (The other three were unavailable from DHSS.) These 34 accidental deaths were coded as follows: 20 natural; 14 accidental. That is, incorrect coding by NCHS accounted for at least 20 of the 37 unrecorded accidental deaths. (And, DHSS problems accounted for at least 14 of the 37 unrecorded accidental deaths. See Section 4.2.2.)

Table 2 shows that NCHS errors occur with alcohol-related deaths especially. It provides details on the 20 coded incorrectly that fall into two groups: alcohol-related (16); non alcohol-related (4). It gives the age and sex of the decedent, the blood alcohol content (bac), the cause-of-death given on the certificates, and the code entered on them by NCHS. The two groups are now discussed separately.

Combined, three sources of information show unambiguously that the 16 individuals died soon after, and because, they had ingested alcohol in large amounts: the troopers' reports which gave background information; the autopsy protocols that included the bac levels; the certificates that enumerated the causes of death. NCHS coded all of them as natural deaths (O-799) in six codes, the most common being the 300 group: 303, alcohol dependence syndrome; 305, non-dependent abuse of drugs. Combined, these two account for 10 of the 16 alcohol-related deaths incorrectly coded as natural.

This pattern indicates a generic problem with the NCHS coding rubric: a certificate that explicitly includes alcohol or alcohol-related symptoms as the causes-of-death appears to steer NCHS into selecting a cause-of-death code within the manner-of-death grouping for natural death: O-799.

One example —number eight— suffices to make the point that coding such deaths as natural is inappropriate. This young woman of 17 was found frozen to death on a riverbank the morning after a night of heavy drinking (bac 236; urine alcohol content, 400).

Coding the four non-alcohol-related deaths as natural is similarly inappropriate. Of them, three were coded 799, i.e. other ill-defined and unknown causes of morbidity and mortality. One was a hunter who left his companions and disappeared. The second was a miner, living alone in his cabin, who disappeared. The third was a young woman who died outdoors distant from the village after an epileptic seizure. The fourth was a young man who asphyxiated, as the certificate has it. (In fact, the young man died from aspiration of gastric contents after drinking. The certificate has no mention of alcohol.)

All 20 cases of incorrect coding by NCHS appear to result from the combination of two factors: a predisposition on the part of NCHS to choose a cause-of-death code within the manner-of-death category "natural" when it has the option of doing so, and inadequate information on the certificates that are its only source.

It seems clear that more information on the certificates would help steer NCHS away from errors in coding. This is now shown by comparing its accuracy with two different kinds of certificates: Certificates of Presumptive Death and Certificates of Death. Its accuracy with Certificates of Presumptive Death is much greater, because they contain more information.

As noted, the authors obtained from DHSS copies of 34 NCHS-coded certificates. Of them, 12 were Certificates of Presumptive Death, and 22 were Certificates of Death. Of the 12 Certificates of Presumptive Death, NCHS coded nine correctly as accidents and coded only three incorrectly as natural. But of the 22 Certificates of Death, NCHS coded only five correctly as accidents, and coded 17 incorrectly as natural. (The three and the 17 constitute the 20 incidents shown in Table 2.)

The explanation for this differential success rate

Table 2. Deaths coded natural

	alcohol-related															not alcohol-related				
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
age/sex	33/f	49/m	25/f	27/f	44/f	35/m	45/m	17/f	42/m	46/f	31/m	32/m	35/f	62/f	33/m	42/m	23/m	49/m	31/f	80/m
bac (mg/dcl)	338	405	125	289	498	701	uk	236	306	506	uk	240	514	260	469	uk	—	—	—	—
cause of death (cert. of death)																				
ac. alc. hepatitis	a																			
ac. alc. intox.								a				b								
ac. alc. overdose	b																			
acute alcoholism		a	b																	
acute pneumonitis						a														
alcohol											b									
alcohol overdose					a				a				b		a					
asphyxiation																	a			
asp. gast. cont.			a	b		b				b			a							
chron. pulm. dis.		b																		
clin. pneumonitis										a										
elevated b.a.c.														b						
epileptic seizures																			b	
exposure																			a	
fatty infilt. liver															b					
focal bronchopneum				a																
heart problem											a									
hyperthermia											c									
hypothermia								b				a		a						
intoxication							b													
possible asp. gc							a													
sev. ac. alc.				c																
undetermined																		a		
unknown																a				a
very drunk																b				
cause-of-death: (NCHS code)	571	303	303	303	305	507	305	305	305	507	305	305	305	790	571	799	799	799	345	799

legend: a,b,c — order in which causes-of-death are given on certificates
bac — blood alcohol content (milligrams per deciliter)
303 — alcohol dependence syndrome
305 — nondependent abuse of drugs
345 — epilepsy
507 — pneumonitis due to liquids and drugs
571 — chronic liver disease
790 — nonspecific abnormal findings on examination of blood
799 — other ill-defined and unknown causes of morbidity and mortality

appears to lie in the greater information provided by the Certificates of Presumptive Death. The differences between the two certificates are now summarized.

Certificates of Presumptive Death, like Certificates of Death, include a space for cause(s)-of-death and boxes for manner-of-death: accident, homicide, and suicide (but not undetermined). Unlike Certificates of Death, they have also a box for violent death and explicitly include space for a summary of evidence.

In all 12 Certificates of Presumptive Death, the cause-of-death was filled in locally. In 11 of the 12, the manner-of-death also was filled in locally—as an accident. In six the box for violent death was also filled in locally. The summary of evidence was included in ten. Only one of the 12 had the boxes for manner-of-death and the box for violent death been left blank, but it included the summary of evidence.

By contrast, 19 of the 22 Certificates of Death had only the cause-of-death, while three had both cause-of-death and manner-of-death (accident) filled in locally.

In the authors' opinion, the manner-of-death was filled in on the Certificates of Presumptive Death because the summary of the evidence compels it, and was not filled in on the certificates of death because they do not require a summary.

Whatever the reason, in nine of the 12 certificates of presumptive death, NCHS coded correctly for accident, i.e. accepting the local determination of manner-of-death. It seems clear that an explicit local determination of manner-of-death and the inclusion of a summary of evidence in a certificate guides NCHS away from coding errors. In sum, most of NCHS coding errors were on certificates of death, and NCHS coded few certificates of presumptive death incorrectly.

4.2.2 DHSS PROBLEMS

As noted (Section 4.2.1), the printout of violent deaths excludes 14 deaths coded correctly by NCHS as accidental. The quote from DHSS included above (Section 4.1.3) may be the explanation. And, it may explain why the printout excludes one suicide (Table 1, line 13) determined as such by the authors and correctly coded by NCHS. This may be an idiosyncratic case. Or it may reflect fundamental problems within DHSS, to whom the authors have referred the case. It is summarized here because it appears to point to fundamental problems within DHSS and because it raises a unique question. Hitherto, the authors have raised the question: How does NCHS come to code incorrectly? In this instance, the question is: How did NCHS come to code correctly?

The death occurred in 1982. The Delayed Certificate of Death was signed by the pathologist one month after the suicide occurred. It was not signed by the local coroner until February 22, 1985. DHSS stamped it received on February 7, 1985. The printout of violent deaths that excluded it was furnished to the authors by DHSS six months later, i.e. November, 1985. The authors do not know why it was excluded.

The authors determined that it was a suicide, although no official document in the coroner's file on the case established it as such. (Pertinent documents are: inquest proceedings; orders to dispense with inquest; affidavits for correction of a record.) The basis for the determination was the trooper's report. It refers to the individual's having: talked about suicide prior to the event, having a "very heavy" drinking problem, and having been intoxicated at the time of death.

The certificate of death is ambiguous. Manner-of-death is left blank. The cause-of-death is given as "gunshot, chest." It could have been an accident, a homicide, or a suicide. NCHS coded it correctly: E9551, i.e. suicide and self-inflicted injury by firearms and explosives.

5.0 SUICIDE

Table 1 (lines 9 and 14) shows that eight suicides were overreported as accidental deaths in the DHSS printout on violent deaths, i.e. underreported as suicides. (And, it shows [line 13] that one was not recorded. This is the incident just discussed.)

The eight underreported suicides are deaths that should (in the opinion of the authors) have been reported as suicides but were not.

The table shows that underreporting (with eight cases, line 14) is much less important than either misrecording (40 cases, line 3) or underrecording (38 cases, line 13).

But underreporting of suicides is likely to continue, whereas in principle the problems of misrecording and underrecording are capable of being resolved.

This section discusses three things: underreporting, the population at risk for suicide in the region, and how its rates compare with rates among Natives in Canada and Greenland.

5.1 Underreporting

Eight of the nine cases determined by the authors to be suicides were coded by NCHS and included in the DHSS printout as accidental deaths. Three were ruled accidents at inquest, three were ruled accidents by coroners in orders to dispense with inquests, one was the subject of an order to dispense with inquest that did not specify the manner-of-death, and in one an order to dispense has not been issued nor an inquest held. The ninth is the case just discussed on which no local determination has been made.

The authors' determinations were based on the information in the coroners' files: troopers' reports and inquest proceedings. The troopers' reports in particular have many of the characteristics of a psychological autopsy even though the troopers have no formal training in psychiatric, psychoanalytical, or psychological techniques. A psychological autopsy is a compilation of facts about the decedent's life gathered in the hope that they may shed some light on its ending: psychological state, interpersonal relations, loss of loved ones, drinking habits, employment status, violence or withdrawal, remarks about self, and others. Such dossiers are then seen as helpful in preventive planning.

The troopers' reports are vital for an understanding of the circumstances and are (or could be) very helpful in health planning. They provided the information required to apply the criteria adopted—explicit and implicit indicators and the overall balance of the evidence. Table 3 shows the criteria used.

In most cases, several criteria were present at the same time. In the three cases where few criteria applied (Cases 3,4, and 9), one had ingested a lethal dose of drugs; the second was acutely intoxicated, while the physical evidence —the position of the body and weapon and the existence of powder burns— was unambiguous; the third had stated his intent prior and had been drinking continuously for some time.

The presence of alcohol is a complicating factor. In one example, the inquest jury delivered a verdict of accidental death by self-inflicted gunshot because of it. This reflects the fact that the coroner/magistrate is required by law to define suicide for the jurors as the "...act of taking one's own life voluntarily and intentionally (8)."

That the decedent was intoxicated at the time is seen by jurors as leaving open the question of whether or not suicide was the decedent's intent. Juries are for several reasons inclined to bring in a verdict of accidental death unless a verdict of suicide is inescapable. These are small villages, and it spares the feelings of the next-of-kin who are neighbors and may be the relatives of some of the jurors. It avoids the discomfort attending refusal of burial in the consecrated ground of the Catholic and Russian Orthodox Churches.

Given their cultural and religious predilections and this legal definition, juries fasten on the degree of intoxication as indicating that the decedent was not able to form the intent to commit suicide. Knowing this, cor-

Table 3. Criteria used in authors' determinations of suicide.

Criteria	Decedent #								
	1	2	3	4	5	6	7	8	9
explicit									
witnessed	x								
physical evidence				x					
previous attempt(s)	x								
stated intent prior	x				x			x	x
stated intent after event							x		
implicit									
signs of									
mental disorder		x				x			
stress		x				x	x		
depression	x	x							
loss of loved one	x	x			x	x	x	x	
extreme alcohol abuse	x	x		x	x	x	x		x
extreme drug abuse		x	x		x				
loss of job		x							

Sources: Coroners' files

oners may elect to issue an order to dispense with inquest, ruling the death an accident. As noted, three of the nine were dealt with in that way.

Since alcohol was involved in two-thirds of the 38 suicides, underreporting is expected to continue.

Underreporting has been widely discussed. In one study McCarthy and Walsh (9) computed that the suicide rate in Dublin over the period 1964-68 was almost four times that reported by the coroners. The differences stemmed partly from coding and partly from the different criteria used: theirs clinical, the coroners' legal. The nine underreported here reflect similar differences, since our clinical criteria included the information gleaned from the files that has many of the characteristics of a psychological autopsy.

Sainsbury and Jenkins (10) concluded that such sources of error do not invalidate epidemiological comparisons of suicide rates among countries and districts within them. The authors do not dispute this, but believe that underreporting of suicides is a consistent and significant hindrance for local health planning which needs reliable absolute numbers.

The psychological autopsy is an important tool in such planning, but appears to be neglected in the region under study. Shneidman and others (11) developed it to help understand the circumstances surrounding suicides in the belief that the standard categories—accidental, natural, suicides, undetermined—do not adequately capture the complexity of deaths from suicide which reflect a continuum of psychological states rather than an either/or contrast. Farberow, Shneidman, and Litman (12) argue that the categories need to be revised to take into consideration conscious and unconscious motivation, suggesting unmeditated, premeditated, and submeditated death. The psychological autopsy permits such more-sophisticated classification: it complements the somatic autopsy by gathering information on the life of the decedent, especially from people who knew him or her, to establish a social and psychological profile. It has developed along preventive and therapeutic lines (Weisman (13)).

5.2 Population at risk

Table 4 shows the characteristics of the 38 suicides. The population most at-risk is single males aged 15-29, who account for 73 percent of the total. Firearms were used in 71 percent of the cases, especially by males, whereas drug overdose was more typical of females. Seven of the nine females were also aged 15-29. Alcohol was involved in two-thirds of the suicides for both males and females. Prior indicators were more prevalent among the males than among the females.

5.3 Rates

All but two of the suicides were Native. Deducting the two non-Natives yields an average annual rate among Natives for the period 1979-84 of 36.9 per 100,000 as compared with an official rate, based on only five suicides, of 7.3 per 100,000. The official Alaska rate for non-Natives was 15.2 in 1984 (14,15). These numbers are consistent with other data showing much higher rates among Natives than among non-Natives and are similar to other recent data. For example, Syer (16) reports official statistics showing that the rate among

Indians and Inuit in Canada was 41.0 per 1000,000 in 1977, as compared with a rate of 12.8 in Canada as a whole in 1976. And Lynge (17), again using official statistics, shows Native rates in Greenland of 50 per 100,000 in 1976 and of 90 per 100,000 in 1982.

6.0 COMMENTS AND CONCLUSIONS

In these small villages where only seven of the 49 in the region have more than 500 people, alcohol-related accidental deaths are a continuing tragedy of enormous proportions. In the subregion the authors lived in for eight years, the combination of such deaths, of homicides, of suicides, and of incarceration of individuals for alcohol-related felonies has reduced the growth in the labor force by 25 percent. This aggregate statistic conceals the effects on individual villages: in one there are few young men left.

No purpose is served by coding such deaths as natural. They are not. Preventive health planning is hampered by such coding: the numbers it yields are worthless.

Some improvement in NCHS coding may be achieved by four things:

1. Change the format of the certificates to enable coroners to provide more relevant information,
2. Train coroners and pathologists in what relevant information is from the point of view of the nosologist,
3. Train and encourage coroners in punctilious observance of the system set up to ensure good communication between them and DHSS,
4. Implement a program that increases the sensitivity of NCHS (and the DHSS nosologist—trainee in Juneau) to what may be the unique nature of the raw data flowing from rural Alaska, so that they will spot more readily certificates that call out for query, and query them.

Table 4. Suicide characteristics

	method								
sex, age & mar. status	**fire-arms**	**od meds**	**hypo+ meds.**	**agc+ meds.**	**drown+ od meds.**	**drown**	**total**	**alc-inv.**	**prior indic.**
males									
single									
15–19	6			1			7	5	5
20–24	7	1	1				9	7	5
25–29	3	1				1	5	5	4
30–34	1						1	1	1
35–39	2						2		2
40–44					1		1		
45–49	1						1		1
married									
55–59	1						1	1	1
65+	1						1		1
widowed									
60–64	1						1	1	
subtotal	23	2	1	1	1	1	29	20	20
females									
single									
15-19		1					1		1
20–24	2	1					3	1	1
25–29	1						1	1	
married									
25–29	1						1	1	
separated									
25–29		1					1	1	
widowed									
50–55				1		1	2	2	1
subtotal	4	3		1		1	9	6	4
total	27	5		2	1	2	38	26	24

Notes: **od meds = overdose of medications**
hypo+meds = hypothermia plus medications
agc+meds = aspiration of gastric contents + medications
alc inv = alcohol involved

Beyond that, it may be that ICD-9 as presently constructed cannot accommodate the kind of accidental deaths reported here in a way that places them squarely in the broad category of accidental death rather than in the broad category of natural death: the young woman of 17 who died sometime during the night on a riverbank after drinking heavily, whose death was coded natural —305, nondependent abuse of drugs.

Local mental health professionals may benefit from establishing good working relations with the troopers, whose dedication and reports are remarkable, and from devising region-specific psychological autopsies for use in preventive health planning.

At one level, the data problems disclosed may reflect the unique circumstances of village life in rural Alaska. But at another level—that of DHSS internal problems —they do not. These internal problems—that show up especially in the non-inclusion in the vital statistics on violent deaths of deaths correctly coded by NCHS as accidental—presumably apply also to urban Alaska data and to non-Natives as well as Natives. The authors have no recommendations on this problem since it appears to reflect data-handling shortcomings on the part of DHSS staff who are, or should be, well-enough trained to overcome them.

The criteria adopted (Table 3) by the authors in determining suicides, despite local documentation to the contrary, are offered as a contribution to the debate. That is, the authors have avoided attempting to weight the criteria adopted or even to establish a minimum number. The overall "balance of the evidence" reflects their sense of what is plausible based on their knowledge of the thoughts, feelings, and behavior of village residents during their eight years in the region, where one of the authors was an alcohol and mental health councilor for six years. Other criteria may be needed and some included may be found wanting. That is for the profession to decide. Table 3 is only an attempt to explicate formally what began and ended as an intuitive approach.

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A Historical Perspective on the Physiology of Carbohydrate Loading

by Jay E. Caldwell, M.D., M.P.H., F.A.C.S.M.(1)

If your medical training was anything like mine, its nutrition content was nil. I do recall trying to memorize which vegetables and grains jungle dwellers were supposed to mix in order to achieve a balanced protein intake, but that's about it.

Understandably, you may feel a bit out of sorts when an athlete presents at your office looking for dietary advice about preparing for a triathlon or a marathon. If you choose to accept the assignment, no doubt the concept of "carbo loading" will arise. This historical overview and the references that follow may provide you some well-needed background.

The story of carbohydrate loading is now well into its third decade. In the early 1960s physiologist Bengt Saltin and his coworkers at the Karolinska Institute in Stockholm began experimentation with the then novel muscle biopsy procedure. In the five-year period following 1966, they published a series of papers (1-5) that today form the foundation for today's concept of carbohydrate loading.

First they noted that when untrained men peddled to exhaustion on a cycle ergometer, their endurance was directly related to the amount of glycogen present in their vastus lateralis muscle before exercise (4). Later, the same effect was seen in well-conditioned men, as well as with itermittent exercise (6).

Assuming that the glycogen had to be of dietary origin, they undertook a series of nutritional manipulations. In one experiment subjects were asked to perform one-legged cycling. They then ate a high carbohydrate diet for three days. Muscle biopsies showed that the exercised leg was restocked with more than twice the glycogen as the unexercised leg (1).

The final paper in the series was the classic. In this experiment the athletes first rode to exhaustion on the ergometer, then went on a 3-day, very low carbohydrate diet, and finally made one more endurance ride. These efforts were directed at emptying the muscle as completely as possible of glycogen, so that it would more readily fill when provided the opportunity. Three days of rest and high carbohydrate intake ended the protocol. It worked. Both muscle glycogen and performance skyrocketed (3).

The religious fervor for carbohydrate loading today is based on these studies. Unfortunately, they have been misinterpreted and misapplied. All these studies involved cycle ergometer exercise, which is a controllable device in the lab but often has little to say about running. These data have now been replicated in swimmers but not in runners. In fact, running seems to delay the refilling of glycogen stores.

In 1966 Bergstrom and Hultman (1) noted that after exhaustive cycling exercise, glycogen stores were refilled within 24 hours, and "supercompensated" after three days. In 1971 Costill (7) reported that following successive days of treadmill running, there was incomplete refilling of glycogen stores, even when the athletes were force-fed a 4,000 calorie, high carbohydrate diet. Serial muscle biopsies suggested that a minimum of 48 hours was necessary to insure adequate glycogen replenishment (8).

Later Sherman et al. (9) reported that following a marathon, glycogen stores were less than two-thirds refilled even after a week of a high carbohydrate intake.

Unfortunately, in the running community the ever present fallacy of "if a little is good, more must be better" had taken hold. Costill (10) noted that following three days of running, a 3-day high carbohydrate diet resulted in a very high level of glycogen deposition. If this were so, the reasoning went, why not really hammer yourself during the depletion stage; then you could really plump up those muscles with carbos.

Burke and Read (11), in a study of Australian runners, found that there was a great discrepancy between the principles espoused by physiologists and nutritionists (12) and the actual practices of marathon runners. Most ate too little and exercised too much prior to important races.

Recently Costill, this time working out of a Norwegian lab (13), has put to bed, permanently, the "run to exhaustion" concept of carbohydrate loading for elite as well as recreational athletes. He observed that over the course of a three-day, tapered running program, a 3,000 calorie (53% carbohydrate) diet induced an increase of muscle glycogen from 110 to 180 mmoles/kg (wet muscle). Two hundred is generally considered a supercompensated glycogen level; presumably a higher percentage of carbohydrate might have achieved this. Interestingly, the glycogen level of untrained runners reached only 102 mmoles/kg.

Next all the subjects ran for a total of an hour and three-quarters on the treadmill at 75% of their max.

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Muscle glycogen plummeted to 26 mmoles/kg in the nonathletes and to 57 mmoles/kg in the athletes. All then ate 3,500 (69% carbohydrates) calories daily for the next three days. After the first day the glycogen rebounded only to 87 and 127 mmoles/kg, respectively, but remained unchanged for the next two days.

Glycogen synthase mediates the deposition of glycogen in intact muscle. A low glycogen concentration is a stimulus for this enzyme. There is no question that exhaustive running depletes fuel stores (14). Unfortunately, there is now clear evidence that running, even moderate running, injures muscle tissue so that its metabolic machinery is temporarily dysfunctional (15).

This muscle injury is akin to the delayed muscle soreness that follows new exercise efforts, and in particular those that include eccentric contractions (16), "negatives," as weightlifters call them. These occur when a muscle lengthens at the same time it develops force, as when it resists the downward pull of gravity. In running the quadriceps makes an eccentric contraction at every footfall. Because swimming and cycling do not involve eccentric contractions, and walking very few, they induce minimal tissue damage.

Morphological changes in mitochondria have been noted in muscle following running, and it is well known that serum levels of muscle enzymes are elevated after distance running (9). It is reasonable to postulate that membrane transport mechanisms and enzyme coupling may be altered, thereby inhibiting resynthesis of glycogen (17).

The message is clear. For the runner to hammer him/herself prior to an important race does no good and is more than likely to be counterproductive. A carefully planned 3–5 day taper, coupled with lots of fluid and carbos, is the best recipe. Because there is a limit to the amount of carbohydate muscle can store (18), 3,500–4,000 calories, with 65–70% coming in the form of simple and complex carbohydrates (600 gms), seems to be the ideal.

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The Alaska WAMI Program: A Preliminary Study of Factors Affecting Specialty Choice and Practice Location

by Sven O. E. Ebbesson, Ph.D., D.Sc.[1]

ABSTRACT

Alaska's participation in the Washington-Alaska-Montana-Idaho (WAMI) Medical Education Program was prompted by the need for direct access to medical education and for more primary care physicians in rural areas. This report is based on data obtained from a questionnaire filled out by 42 graduates of the program who have completed their residency training. Of the alumni in Alaska, 52% are currently practicing in small towns, and of these, 91% are in family practice. Since the national average for medical school graduates to enter primary care practice in rural areas is only 8%, the WAMI Program is exceptionally successful in turning out physicians for small communities. The extensive exposure to medical practice in rural Alaska during the first and third year of medical school played an important role in decisions about specialty choice and practice location. More alumni would return to Alaska and the rural areas if the number of clerkships offered in Alaska were increased and if residency programs were established in Alaska.

INTRODUCTION

The Alaska WAMI Medical Education Program is now 15 years old, and the first alumni have now returned to Alaska after completing their training. This report deals with an analysis of the distribution and specialty chosen by the alumni and the factors that contributed to their decisions about specialty choice and practice location. The study was undertaken in order to evaluate the program and to determine if the goals defined 15 years ago are being achieved.

When the WAMI Program was conceived, Alaska was one of the few states without direct access to medical education. Any aspiring premedical student had to find his or her own way and obtain admission to a medical school in the lower 48 and secure financing for the education. Not only was it difficult to gain entrance to private medical schools but also the expense was beyond many. Most state schools take no or very few out-of-state residents. Coupled with the need for student access to medical school was a desperate need for more primary care physicians in the small Alaskan communities. The conception of the founders of the WAMI program was that almost all Alaskan physicians were located in the larger cities and too few served the small communities. This need was one of the main considerations when the University of Alaska entered into a partnership with the University of Washington, Washington State University, Montana State University, and University of Idaho to form the WAMI Program. The AMI partners were essentially in the same boat as Alaska in that they needed access to medical education without being independently able to build their own medical schools because of high cost and relatively small number of potential medical students. The WAMI program thus came into being to 1) provide access to medical education, 2) increase the number of primary care physicians in rural areas of all four states, and 3) to accomplish this without capital expenditures in the AMI states.

The program initially was funded by a $1 million grant from the Commonwealth Fund of New York and later by the federal government ($8.1 million). Alaska was the first of the AMI states to admit students in 1971. A total of 15 students were admitted 1971-1974, but when the Alaskan legislature began to provide funding in 1975, 10 students a year were admitted. In 1984, enrollment was increased to 13, and in 1985 the number was increased further to 15. Because of declining oil revenues, the state reduced the number of students to 10 each in 1986 and 1987. Beginning with 1987, students go directly to Seattle (or Montana and Idaho) for the first year instead of to Fairbanks.

To attract WAMI students to the Alaskan rural areas, a special effort was made to expose the students to opportunities in small towns and to the joys of primary care practice. During the first year in Fairbanks, students have the opportunity to take two preceptorships as electives, each composed of spending half-a-day a week for six weeks with carefully selected practitioners. In the spring semester the students have another elective in "rural health" which includes a 7-9 day field trip to rural Alaska where they have an opportunity to work, and often to live, with a local practitioner. Typical sites for such trips are Kotzebue, Nome, Dillingham, and

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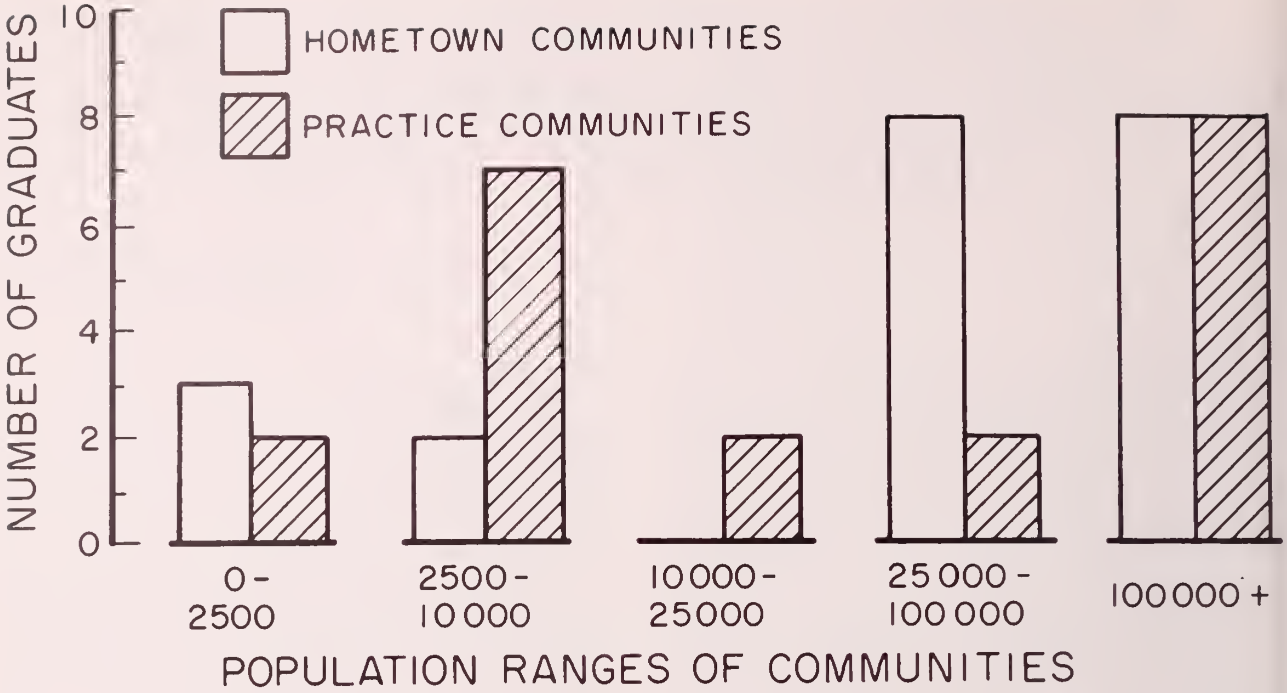

Figure 1. Distribution of Alaskan WAMI graduates practicing in Alaska by size of hometown versus practice location.

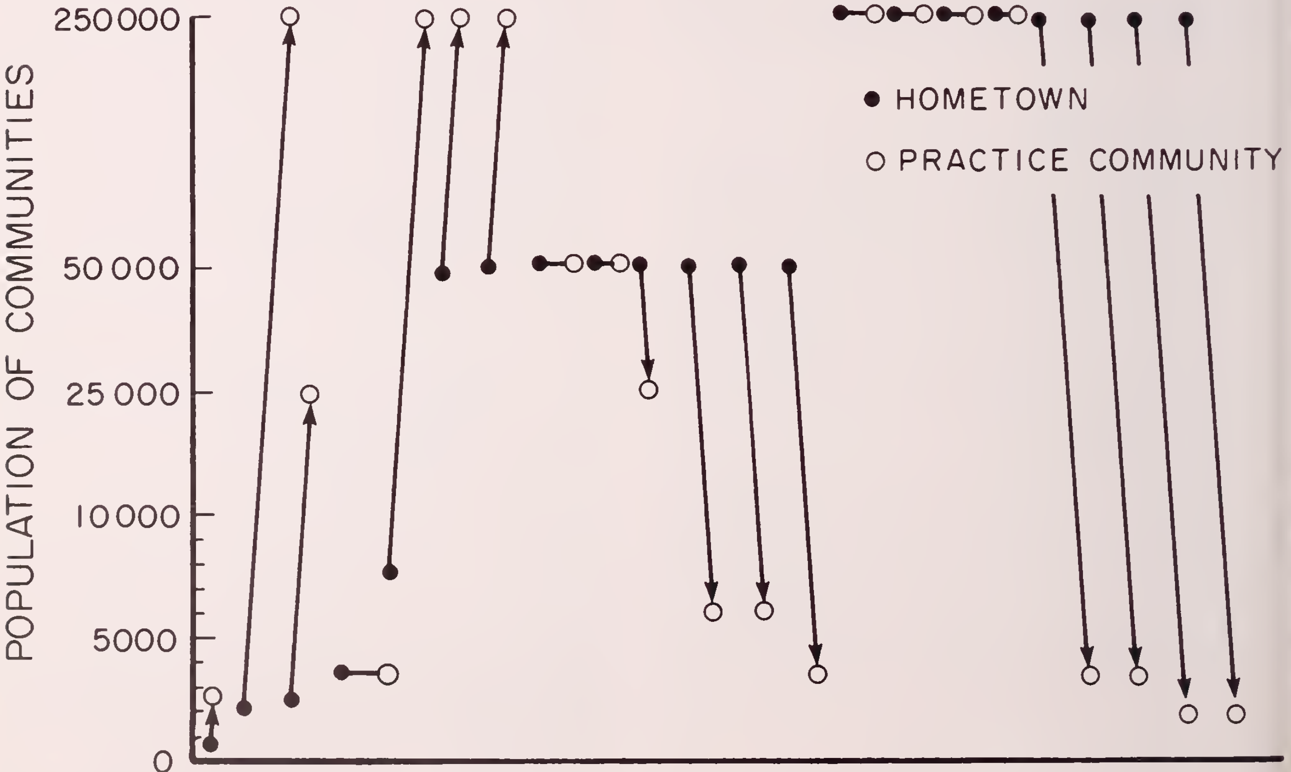

Figure 2. Distribution of individual graduates by size of hometown versus practice location.

Barrow. Almost all students take these electives and respond enthusiastically to the experience. The second year is spent in Seattle, but during the third and fourth years, the students have opportunities to take clerkships at various sites in the four state area. The University of Washington offers four clerkships in Alaska: family medicine in Ketchikan and Anchorage (Alaska Native Medical Center), psychiatry in Anchorage, and OB/GYN in Anchorage. Field trips to small communities also are part of the clerkships. Additional clerkships in such places as Bethel and Fairbanks are also popular with the students. Some 92 Alaskan physicians are now involved in teaching medical students.

The question now is: Do these Alaskan experiences play a role in decisions about specialty choice and place of practice? Teleologically one would predict so. But we know from other studies that many other factors are powerful determinants of both specialty choice and practice location. For example, many set up practice near the location of their residency (1). Other factors include the wish or job opportunity of the spouse, opportunity for partnership, climate or geography, and income potential (1,2). It is also known that most physicians reared in metropolitan areas settle in similar sized communities. At the University of Oklahoma Tulsa Medical College, 71% of those graduates who were reared in communities of 100,000 or more showed a preference for practice locations in communities of that size (2). A study of first and second year WAMI students showed the same trend in that the students from metropolitan areas, in general, planned to practice in similar sized communities (3).

Given the small number of WAMI alumni who have had time to return to Alaska and the uniqueness of Alaska, its population and population distribution as compared to the rest of the United States, the survey reported here provides only preliminary results that can only tentatively be related to other studies.

METHOD

In September 1986, a questionnaire was sent to all graduates who finished their residencies to determine what factors they considered important in selecting their practice location and specialty. Information also was obtained about the size of community presently served, net income, schools attended, and specialty training.

Graduates were asked if the preceptorships during the first year in Fairbanks contributed to decisions about (a) specialty choice, and (b) returning to Alaska. They were asked about what clerkships were taken in Alaska and if the clerkship experience in Alaska contributed to specialty choice. The survey asked the graduates if they could have afforded to attend medical school if the WAMI program had not existed and how much money they had to borrow for medical school.

RESULTS

Of the 47 graduates in practice, 43 (91%) responded to the questionnaire, and of these, 19 (90%) of the 21 practicing in Alaska responded. Of the 26 graduates practicing outside Alaska, 24 (92%) returned their questionnaires. We have not included the information from 5 physicians practicing in the military or in the public health service abroad. Although some references will be made to our findings about Alaska WAMI alumni practicing outside Alaska, the present discussion will deal primarily with alumni practicing in Alaska. A summary of the findings is presented in Table 1.

DISCUSSION

Although approximately 40% of Alaska's population is distributed outside Anchorage and Fairbanks, 52% of the WAMI alumni are practicing in small towns. This means that the goals of the architects of the program are being reached. The proportion of graduates ending up in small towns is high compared to other programs in the United States. A study of Oklahoma graduates, for example, showed that 16% ended up in towns of less then 25,000 people (2).

The questions are: What factors contribute to practice location, and why is such a high proportion of WAMI graduates going to non-metropolitan areas? A number of factors have been mentioned above. These include: size of hometown, location of residency program, opportunities for partnership, job opportunity for spouse, location of family, income potential and climate (1–3).

The role of size of hometown.

A comparison of size of hometown with the size of practice location of the Alaska WAMI graduates reveals a shift from cities to small towns (Figure 1). An analysis of individual cases shows that 53% of the WAMI graduates that came from cities are now practicing medicine in small towns (Figure 2). Although the numbers are small in the present study, the proportion of graduates moving from a city to a small town is considerably greater than in other populations of physicians so far examined.

For example, Holmes and Miller (2) noted a strong preference (71%) for establishing practice in cities if the physician grew up in such communities. Bible (4) and Champion and Olsen (5) also noted this trend. Carline and coworkers (3) found a similar trend when asking first and second year WAMI students where they intended to practice medicine. They suggested, "An admission program aimed at increasing the number of students interested in practice in rural areas should include provisions for increasing the number of students raised in rural areas who are admitted to its program."

The present study cannot dispute this argument be-

Table 1. Data on alumni of the Alaska WAMI Medical Education Program and their responses to questions about factors determining their choice of practice location and specialty.

	Practitioners in Alaskan Small Towns[1] (2,000-25,000)	Practitioners in Alaskan Cities (50,000-250,000)	Practitioners in Alaska	Practitioners in Other States
1. Practice location of alumni in Alaska[2,3]	11 (52%)	10[4] (48%)	21[5]	26
2. Specialty[3]				
a) Family practice	10 (48%)	4 (19%)	14 (67%)	
b) Internal medicine	0	3 (14%)	3 (14%)	
c) Pediatrics	0	1 (5%)	1 (5%)	
d) Psychiatry	0	1 (5%)	1 (5%)	
e) Anesthesia	1 (5%)	1 (5%)	2(10%)	
f) Total primary care	10 (48%)	8 (38%)	18 (86%)	
3. Number of alumni with more than 3 years of specialty training	1	1	2	7
4. Board certified	82%	75%	79%	71%
5. Born in Alaska	18%	50%	32%	14%
6. Attended high school in Alaska	45%	88%	63%	58%
7. Attended college in Alaska	36%	25%	32%	29%
8. Borrowed money for medical studies	64%	88%	74%	67%
9. Average amount borrowed	$11,345	$12,375	$11,779	$9,854
10. Could not have afforded medical school had WAMI not existed	36%	13%	26%	8%
11. Net earnings/yr. —				
a) less than $50,000/yr.	36%	25%	32%	25%
b) less than $75,000/yr.	82%	25%	58%	83%
c) less than $100,000/yr.	91%	63%	79%	92%
d) more than $100,000/yr.	9%	37%	21%	8%
12. Recorded that the experience of the first year in Fairbanks contributed to a desire to return to Alaska	64%	63%	64%	54%
13. Recorded that the preceptorships during the first year contributed to decisions about:				
a) specialty choice	73%	43%	61%	43%
b) returning to Alaska[6]	43%	57%	50%	10%
14. Average number of clerkships taken in Alaska	1.9	1.5	1.7	1.25
Took more than one clerkship	55%	33%	48%	29%
15. Consider the clerkships in Alaska:				
a) better	88%	33%	64%	44%
b) equal	12%	50%	29%	50%
c) worse		17%	7%	6%
than those offered in Seattle				
16. Had more than one Alaskan clerkship who thought it:				
a) better	100%	66%		71%
b) equal		33%		29%
c) worse				
than those offered in Seattle				
17. Indicated that the clerkship experience in Alaska contributed to their choice of specialty	63%	43%	53%	28%

[1] This includes: Dillingham, Seward, Homer, Kodiak, Bethel, Kenai, Palmer, Juneau, and Wasilla.

[2] This does not include an approximately equal number of WAMI alumni that originated in other WAMI states.

[3] This statistic does not include one alumnus who took the first year in Fairbanks but who originated from another WAMI state.

[4] This includes two physicians working at the Alaska Native Health Center.

[5] This does not include five physicians in the military service or those serving overseas in Public Health. It does include one alumnus that took the the first year in Fairbanks but who originated from another WAMI state.

[6] Several graduates indicated "no" but stated they were already decided on returning to Alaska.

cause of the small numbers, and 2 out of 5 graduates that originated in small towns ended up in cities. A tentative but highly suggestive conclusion is that the special effort to expose the students to rural practice in the first, third and fourth years has indeed resulted in a significant alteration in attitude toward practice in rural Alaska.

One graduate that grew up in Anchorage and who is now practicing in a small town volunteered that he "did decide to go to western Alaska as a result of a rural field trip." Others who are new practicing in small towns made the following comments about the first year in Alaska:

- "It gave me a taste for how pleasant low density population areas can be,"
- "I got a feeling of confidence about Bush practice from the rural field trip,"
- "I got to see more of the state and got to see more of Alaskan medical scene," and
- "I would never have considered practicing medicine in Alaska had it not been for the outstanding experience I had during my first year of medical school."

Several factors related to choosing rural practice emerge from the study. The exposure to rural practice is an eye-opener to many and occurs at a time early in medical school when the student is very receptive to suggestions. It may very well be that the impressions gained create images that remain throughout medical school and residency training. Some obviously identify role models and develop lasting bonds with preceptors. One can predict that the reinforcement of bonds, by the student spending as much time as possible during clerkships and residency in rural Alaska, improves the odds of attracting the graduate to rural Alaska.

The need for local residency programs.

This study and others (1,2) suggest the need for local residency programs in primary care fields because of the tendency for practitioners to settle near the location of the residency. If residency programs include experiences in rural Alaska, we could further enhance the probability of placing a reasonable proportion of graduates in rural areas. The residents in turn could enhance the clerkships and provide stimulation for local practitioners.

Information provided by the present study suggests the need for residency programs in Alaska. Several graduates volunteered that such programs would increase return to Alaska. Perhaps, significantly, there appears to be an inverse correlation between length of residency and return to Alaska (see item 3, Table 1).

This is likely due to such factors as losing contact with Alaska, lack of professional opportunities in Alaska, or increased probability of finding a non-Alaskan marriage partner who does not want to live in Alaska—three individuals practicing outside Alaska have not been able to find a suitable practice location in Alaska, 11 have not returned because their marriage partner would or could not move because of family considerations or career opportunities, and one has not found an opportunity for research in Alaska.

Specialty choice.

Eighty-six percent of the Alaska WAMI graduates work in primary care with a slightly higher percentage in small towns (91%, see Table 1, item 2). It is noteworthy that surgical specialties were not selected by this group. Not surprisingly, graduates with training in psychiatry, pediatrics, and anesthesiology were found in the cities. The figure of 86% primary care physicians is somewhat higher than that noted in Oklahoma (73%)(2). Perhaps the early exposure and emphasis on primary care in the WAMI Program is responsible for the difference.

Our findings suggest that exposure to primary care and rural medicine in Alaska contributed to decisions about specialty choice (Table 1, items 13a, 17). The Alaskan experience was more important to those destined for small towns in Alaska than to those going to the cities or outside Alaska. This may be related, partially, to the amount of time spent in clerkships in Alaska, because those destined for the small towns in Alaska on the average took more clerkships in Alaska (Table 1, item 14), and they also rated the Alaskan clerkships better than those who took fewer clerkships and ended up practicing in cities or outside Alaska (Table 1, items 15–16). Perhaps the moral here is that an increased number of clerkships in Alaska and time spent in Alaska results in a greater percentage of graduates entering primary care in Alaska.

Various respondents to the questionnaires provided the following unsolicited comments in relation to their first year preceptorships:

- "I had a particularly admirable family practitioner at the Tanana Valley Clinic during my preceptorship,"
- "I enjoyed Alaskan medicine,"
- "The WAMI preceptorships offer an excellent opportunity for exposure to Alaska's unique situations...."

In relation to their evaluation of the clerkships, the graduates noted that the clerkships in Alaska were better than in Seattle because:

- "They provided a wider variety of experience—more personalized—more hands-on experience than Seattle,"
- "Doc's more in touch with real practice; more primary care oriented,"

- "More one-on-one teaching, better clinical exposure, more patients, more problems—less zebras."
- "More individual attention, closer relationship with preceptor,"
- "More individual physician attention, more interesting patients; unique Alaskan situations (i.e., bush clinics),"
- "These rotations gave me an understanding of some of Alaska's state and federal facilities to which I now almost daily refer/consult. I got to know many physicians with whom I now interact professionally. In addition, because there are fewer students here, I got more responsibility and therefore learned more than in Seattle."

Some noted other factors were pivotal in decisions about specialty choice. One graduate mentioned that his father and brother are family doctors, and two individuals were turned on by professors in Seattle and entered the professor's specialty.

Finances.

About three-quarters of the graduates reported that they had to borrow money for their medical education, although tuition is low compared to most medical schools (Table 1, items 8–9). An interesting figure that emerged (Table 1, item 10) was that 36% of the alumni in small Alaskan towns stated that they would not have been able to afford medical school if the WAMI program had not existed. They are also, with one exception, in the two lowest income groups.

The net income of the WAMI graduates appears to relate to the size of the community (Table 1, item 11). Only 9% of the physicians in small Alaskan towns earn more then $100,000, whereas 37% of those practicing in the city do. On the other end, 82% in the small towns earn less than $75,000 per year, whereas only 25% in the city are in this category. There are several morals here. It appears that those without funds for medical school do not generally aspire to high paying practices. Since these physicians are often interested in rural practice, where incomes tend to be low, large student loans are, in general, incompatible with choosing rural practice. Hence, subsidized medical education must continue if Alaska's rural population is to be served. There are those that subscribe to indentured service in rural areas, but that does not seem fair to the population at large or to the physician. A forced two-year stint in a small town, for example, creates an unhappy situation for the patient who prefers continuity with the same physician year after year.

CONCLUSION

The WAMI Program is achieving its major objective of raising the number of physicians selecting rural primary care practice. Exposure to Alaskan medical practice during the first, third and fourth year of medical school appears to affect the number of graduates that choose Alaskan rural communities for practice. There appears to be a positive correlation between the amount of time spent on Alaskan clerkships and the number choosing practice in rural Alaska.

ACKNOWLEDGMENTS

The author wants to thank the WAMI alumni for promptly providing data for this study. I also want to thank Wayne Myers, M.D.,for encouragement to carry out this investigation and for his valuable advice in interpreting the data. Population figures were obtained from the Department of Health and Social Services.

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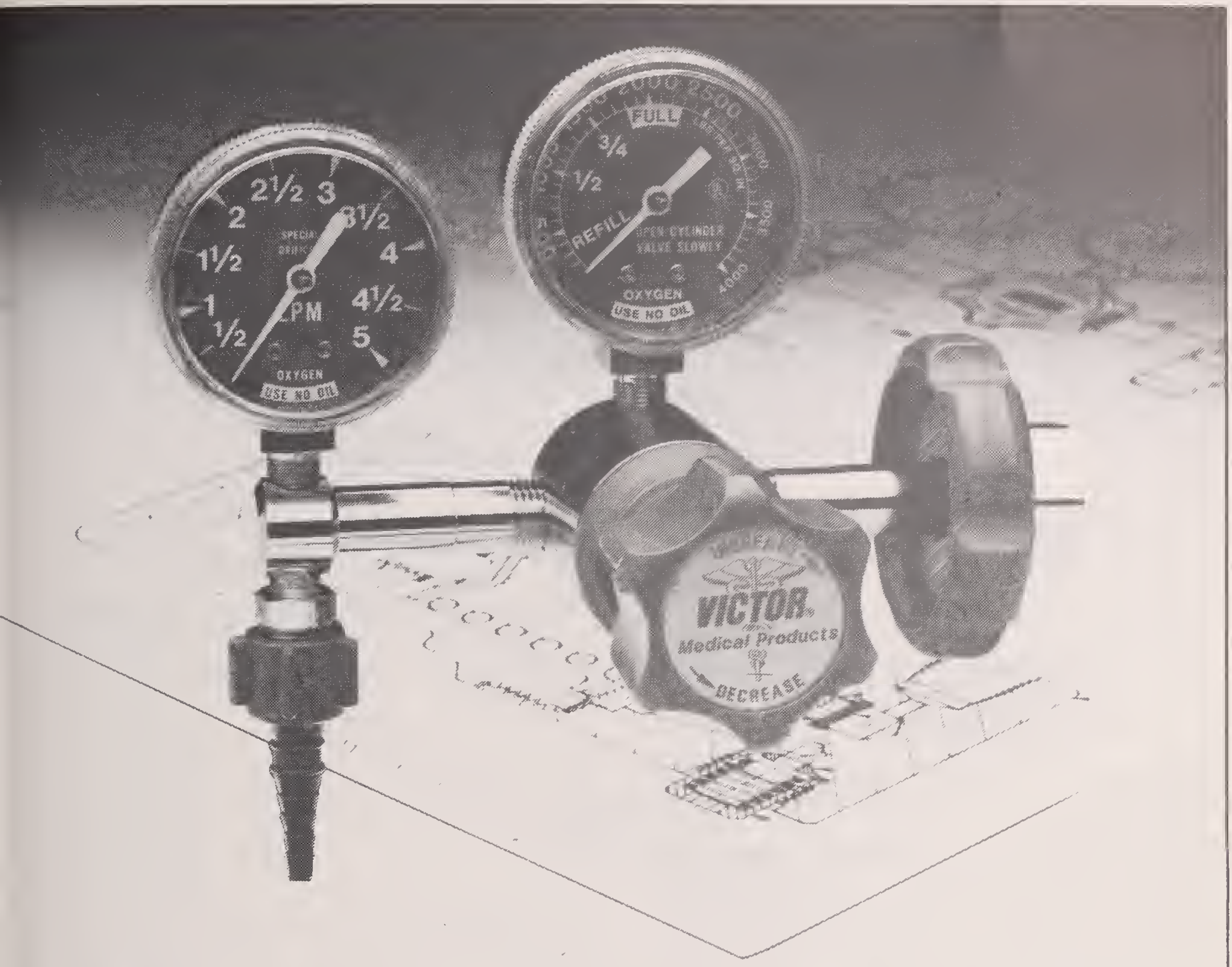

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History of Medicine in Alaska

GLORIA LOREE KNOWLES PARK, M.D.

Gloria Knowles was born in Spokane, Washington but grew up in the Yakima Valley in the small town of Grandview. Her parents "scratched all their lives." They had learned how to manage so well that Gloria "didn't know we were poor."

Her father was reared on a dry farm. Many of the valleys in eastern Washington were virtually desert with sage brush the chief covering. Irrigation was to make a tremendous difference in farming. However, during the 1920s farmers fended as they were able. Gloria's father worked on wheat ranches in the Palouse country and on farms in the Yakima valley during the depression for 50 cents a day. Since he was one of many children, he left home as soon as possible. Eventually, he worked for the Yakima county road department as a mechanic. He regretted that he only had finished the 4th grade and took correspondence courses in order to improve his education and to be able to read manuals. He had natural musical ability and played the violin by ear. Gloria was to master both the piano and violin, but she had to learn to read music.

Her mother was originally from the Dakotas. In 1912, her family had moved west because of drought. Gloria's grandfather worked as an itinerant farm worker but could never accumulate enough money to buy his own farm. Gloria's mother, however, did finish high school and then worked as a clerk for a few years. At this time it was an unusual thing for a young woman to do.

Her parents had one son. Since it was inadvisable for her mother to have more children, Gloria was adopted from the Washington Children's Home in Spokane. She waited until she was fifty before attempting to find her biological parents. She was prompted to do so with the birth of her own first grandchild, feeling that it was useful to have a genetic history and fulfilling a long-standing curiosity.

She found her mother married to her biological uncle. She discovered that she has 5 half-siblings who are also half-cousins. She is looking forward to meeting some of them this spring while traveling in the western states.

Gloria was amused to note that the gap between her front teeth was present in her aunt but not her mother who explained that one of the satisfactions of false teeth was the elimination of the gap.

Gloria was intensely busy even as a child. She recalls that her maternal grandparents were not well. Her grandfather was blind; her grandmother confined to a wheel chair. They lived just a few houses up the street. However, their back-door was visible from Gloria's home. Since there were no phones, this enabled her grandmother to put up a white towel in an emergency. Often Gloria would spend the night in her grandparent's house so they would not be alone.

Grandview was a small town. Her bicycle was a pleasure, but reading was an overwhelming joy. There was a library. In her eagerness, she found that there were books she wasn't allowed to check out; notably "Brave New World." She decided at about the age of ten that she wanted to be a doctor. Then she began to read about physicians, remembering in particular the two volumes about the Drs. Mayo.

Both her brother and she were good students: valedictorians of their classes. Gloria worked summers cutting asparagus and picking cherries. One summer she worked in a physicians's office which cemented her desire to study medicine.

With the money she had earned and the assistance of minor scholarships, she entered Washington State College, now University, at Pullman. To stay in school, she waited on tables and on weekends served as a nurse's aid in the infirmary. After three years, she entered medical school. She applied because she knew she could save some money and because her pre-med counsellor suggested that she should do so. It turned out that he was so well thought of at the University of Washington Medical School that they accepted anyone he nominated. Gloria was to transfer credits back to Washington State, receiving a B.S. in zoology, cum laude, in 1952.

While at Pullman she met Orlo E. Park. Orlo had earned a B.S. in physics and a second degree in education at Washington State. He worked first in cryogenics in Washington, D.C.. When he was transferred to Boulder, Colorado, he discovered that, as his wife, Gloria could attend the University of Colorado on the residency fee. So they were married to live busily ever after.

After being graduated from medical school, Gloria had a rotating internship at St. Anthony Hospital in Denver. Before completing this, son Kevin was born. Fortunately, Orlo's work enabled him to be at home some to take care of the baby, and they had a good landlord, a woman, who lent a hand. Even so, Gloria remembers taking the baby to the hospital when on call. Other physicians in retrospect must have found this appearance of an infant a harbinger of things as yet unknown which were to come. The infant is now a physician.

The next year she took time out from professional work in order to have a second son, Loren. Somehow, with a newborn and a thirteen month old, she looked for a permanent professional job. Orlo and Gloria had decided that it would be better for Gloria to find a position because with his background, Orlo could find a job wherever she went. She wrote to Alaska for possibilities. One of the people she contacted was Bob Wilkins. She secured a contract with the Native Health Service Hospital in Anchorage as a civil service appointee.

Orlo drove the highway, Gloria and sons flew to Anchorage. On arrival, Orlo was to find employment at the liquid oxygen plant on Elmendorf Air Force Base. Later he worked at the coal testing laboratory and finally at the power plant in water testing. Thus he became more of a chemist than a physicist.

Gloria had been hired as a general medical officer. The hospital had been built as a TB sanitorium. There were 500 patients suffering from tuberculosis, but afflicted with a myriad of other diseases as well. There was a staff of thirteen, but, at the time, there was no full time outpatient department. Gloria was placed in the out patient department temporarily though she wanted to do surgery. But now, "She is sure glad she didn't." She has enjoyed a long and great career in ambulatory care, both family and administrative medicine.

Life in the outpatient department was to prove demanding. For one thing it included delivering babies. The hospital was not designed for obstetrics. The mothers were largely from the patients in the sanitorium.

In addition, there were the transient patients. They came by the plane load—either patients going outside for treatment or those returning after two or three years outside. These patients were initially not numbered as statistics nor admitted. They merely used the wards as dormitories. There could be between 40 and 60 patients on one of these flights. Often they had medical needs.

The distribution of medicine was another problem. Patients in transit lost prescriptions or needed refills. In addition, the hospital was the center for the state and all the villages except those in southeast. Patients had to write in for refills and at the same time they wrote about medical problems, asking for diagnosis and treatment by mail. Gloria claims that this was "catalogue medicine": the Sears method.

She was also in charge of Radio Call which was cumbersome. Radio from the Bush was patched through a telephone to another radio or phone and by this means, diagnosis was made and treatment suggested. It was difficult because not all villages had trained personnel of any kind. Often, a reluctant school teacher had to cover this need.

She remembers night call. One person was assigned for the whole hospital. It was hard to finish all the work during the daytime so the doctor on call spent three to four hours making rounds. Except for the pathologist, radiologist and anesthetists, they were on call every third night.

She has vivid recollections of the energy and mischief of the children most of whom were up and about. She felt as if there were an "accident of the day" on the pediatrics ward.

In addition, she was in charge of all the pharmacists, dentists and sanitarians who traveled to the villages.

Her peak load was in 1958/1959 when on one day she saw 165 patients. The only way she could handle such overwhelming days was to have a nurse screen patients. In a flu epidemic, it was necessary to check whole families by finding the sickest to treat personally and by handing those with no secondary infections back to the nurse.

The hospital was slow in changing from a sanitorium. But increasingly as drugs such as INH became effective and as patients began to appear with other diseases and injuries, the hospital gradually broadened its mission. First it added general medicine, then it added specialists. At first physicians in the community were used as consultants; gradually the PHS acquired specialists. Eventually the last TB ward was closed. By then the outpatient department had grown to 10 and the staff to 56.

During the late 1950's and early 1960's, Gloria worked on a system of training health aids for the Bush. Later a program was started with paid health aids who were brought into Anchorage to be trained. With Dr. Dave Dixon, she wrote a Health Aid Compendium.

A field program was begun. Villages were assigned to physicians who would visit them once or twice a year. There were 10 to 12 villages per physician. Service units were set up with the bush hospitals. The physicians stationed at these places were responsible also for the area around them.

Anchorage served as headquarters and had its own service area which actually covered 100,000 square miles. The farthest village, Atka, was 1,200 miles from Anchorage and to get there involved flying to Adak and then a 12-hour boat trip. The boat was a Navy seagoing tug with a round bottom. Seasickness was an inevitable hazard.

Early field trips involved fishing boats, dog sleds, snow machines, and planes of all sizes. Gloria's first ride on a "Goose" was fascinating to her since she didn't know prior to its landing that it could do so in the water. Even more exciting was holding the door of a small plane open when landing on glare ice to slow it down.

Gloria likes the villages. She reminisces about Kokhanok, in the Iliamna area, which was exceedingly primitive when she first went there. The health and economy were both poor. There was nothing: neither clinic, equipment, nor electricity. The visiting staff learned to carry footlockers with necessary equipment. It is unforgettable to hear her tell of doing pap smears with the patient on a studio couch, a coleman lantern for light, and

Gloria sitting on the floor.

On Kodiak, at Ouzinkie, there was a room converted into a chapel for mission use. She remembers screening off space before the altar to run the clinic.

Gloria is impressed by the number of women medical personnel in the public health service in Alaska. At one time there were more women than men on the staff of the hospital. She remembers Ruth Coffin; Beth Coffin (the Coffins were not related); Geraldine Morrow, a dentist; Jean Persons; Florence Styvesant; Ollie Dunn; Lousie Ormand; and Martha Wilson (the last three are dead).

Considering her involvement with the PHS as well as her family which by 1960 had increased to four children, it is remarkable that she has done committee work through the years with the Anchorage Medical Society, Alaska State Medical Association, and Alaska Academy of Family Physicians and has served two terms on the ASMA Council. She also has found time to serve on the Municipal Library Advisory Board.

Gloria thoroughly enjoys a family practice because it does a little of everything. She senses that the villages are families, the perfect place for family practice. Gloria has retired recently from this extremely rewarding practice in order to travel the USA with Orlo. However, because she misses her patients, she volunteers to work between trips. She plans to maintain her professional activities as long as possible.

Just to look at this unassuming woman physician is not to recognize a liberated woman. Through hard work, she has achieved the best, both professionally and personally. It is clear that she has been fortunate in her husband who has supported her as a physician and helped with their family while pursuing his own interests. They both have accepted the hard work making these things possible which says something special about both Orlo and Gloria.

Gwynneth Gminder Wilson
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PROGRAM

Monday, April 25

7:30	Registration Health Fair & Exhibits (all 3 days)
8:30	Introductions
8:40	Welcome (Gov. Cowper)
9:00	Keynote Address (Gov. Matheson)
10:30	Alaska's Health Agenda (Comm. Munson)
Noon	Lunch/AK Diatetic Assoc. Luncheon
1:30	The Public in Health Planning: A Voice to be Heard (Panel) Moderator: Elizabeth Ward
2:45	Concurrent Sessions: A. Getting the Message Across: Effective Public Health Ed. Doris Derelian B. Presentation of Papers
5:00	Adjourn
7:00	Hunger & the Homeless Panel Moderator: Denny DeGross

Tuesday, April 26

8:30	Annual Meeting/Resolutions Continental Breakfast
11:30	Lunch/ Speaker: Mayor Fink
1:30	Injury Control Bob McKnight & John Manning
2:45	Concurrent Sessions: A. Presentation of Papers B. Presentation of Papers
5:00	Adjourn

Wednesday, April 27

8:30	Better Use of Existing Health Data: Focus on Maternal & Child Health Ray Yip
9:45	Concurrent Sessions: A. AIDS: AK Status (panel) Moderator: S.A. Jenkerson B. Presentation of Papers
Noon	Awards Luncheon Moderator: Bert Hall

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Steve Cowper, Governor of Alaska
Doris Derelian, MS, RD, Educator, Nutrition Consultant
Tom Fink, Mayor of Anchorage
Bert Hall, Director, Municipality of Anchorage DHSS
Sue Ann Jenkerson, RN, Epidemiology Section, Div./Public Health
John T. Manning, MPH, EMS, Div./Public Health, AK DHSS
Bob McKnight, ScD, Interim Director, School of Health Sc., UAA
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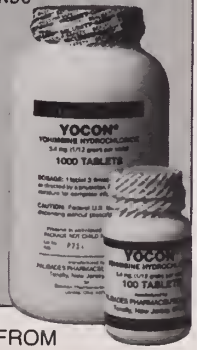

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Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium

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K-DUR tablets contain micro-crystalloids which disperse upon disintegration of the tablet These micro-crystalloids are formulated to provide a controlled release of potassium chloride The dispersibility of the micro-crystalloids and the controlled release of ions from them are intended to minimize the possibility of a high local concentration near the gastrointestinal mucosa and the ability of the KCl to cause stenosis or ulceration. Other means of accomplishing this (e.g , incorporation of potassium chloride into a wax matrix) have reduced the frequency of such lesions to less than one per 100,000 patient years (compared to 40–50 per 100,000 patient years with enteric-coated potassium chloride) but have not eliminated them The frequency of GI lesions with K-OUR tablets is, at present, unknown. K-OUR tablets should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs

Metabolic Acidosis—Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, potassium acetate, or potassium gluconate.

PRECAUTIONS: The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis per se can produce hypokalemia in the absence of a deficit in total body potassium while acute acidosis per se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease, or acidosis requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the electrocardiogram, and the clinical status of the patient

Laboratory Tests: Regular serum potassium determinations are recommended In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis

Drug Interactions: Potassium-sparing diuretics, see **WARNINGS.**

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term carcinogenicity studies in animals have not been performed

Pregnancy Category C: Animal reproduction studies have not been conducted with K-DUR It is also not known whether K-DUR can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. K-OUR should be given to a pregnant woman only if clearly needed.

Nursing Mothers: The normal potassium ion content of human milk is about 13 mEq per liter Since oral potassium becomes part of the body potassium pool, so long as body potassium is not excessive, the contribution of potassium chloride supplementation should have little or no effect on the level in human milk

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS,** and **OVERDOSAGE).** There have also been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS**); other factors known to be associated with such conditions were present in many of these patients

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose

Skin rash has been reported rarely.

OVERDOSAGE: The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic electrocardiographic changes (peaking of T-waves, loss of P-waves, depression of S-T segment, and prolongation of the QT-interval). Late manifestations include muscle-paralysis and cardiovascular collapse from cardiac arrest

Treatment measures for hyperkalemia include the following:

1 Elimination of foods and medications containing potassium and of potassium-sparing diuretics.

2 Intravenous administration of 300 to 500 ml/hr of 10% dextrose solution containing 10–20 units of insulin per 1,000 ml

3 Correction of acidosis, if present, with intravenous sodium bicarbonate

4 Use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia, it should be recalled that in patients who have been stabilized on digitalis too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

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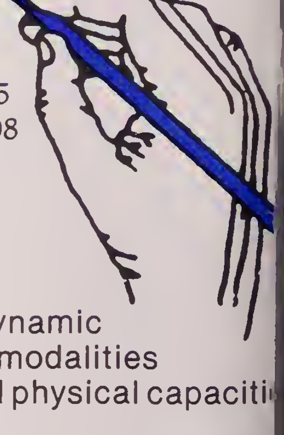

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Before prescribing, see complete prescribing information in SK&F LAB CO. literature or PDR. The following is a brief summary.

Contraindications: There are no known contraindications to the use of 'Tagamet'.

Precautions: While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitorng of prothrombin time is recommended, and adustment of the anticoagulant dose may be necesary when 'Tagamet' is administered concomitantly. nteraction with phenytoin, lidocaine and theophyline has also been reported to produce adverse clinial effects.

However, a crossover study in healthy subjects reeiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. .s. concomitantly with a 300 mg. b.i.d. dosage of heophylline (Theo-Dur®, Key Pharmaceuticals, Inc.), demonstrated less alteration in steady-state theophylline peak serum levels with the 800 mg. h.s. regimen, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly unlikely. A single case of biopsy-proven periportal hepatic fibrosis in a patient receiving 'Tagamet' has been reported.

How Supplied: Tablets: 200 mg. tablets in bottles of 100; 300 mg. tablets in bottles of 100 and Single Unit Packages of 100 (intended for institutional use only); 400 mg. tablets in bottles of 60 and Single Unit Packages of 100 (intended for institutional use only), and 800 mg. Tiltab® tablets in bottles of 30 and Single Unit Packages of 100 (intended for institutional use only).

Liquid: 300 mg./5 ml., in 8 fl. oz. (237 ml.) amber glass bottles and in single-dose units (300 mg./5 ml.), in packages of 10 (intended for institutional use only).

Injection:
Vials: 300 mg./2 ml. in single-dose vials, in packages of 10 and 30, and in 8 ml. multiple-dose vials, in packages of 10 and 25.

Prefilled Syringes: 300 mg./2 ml. in single-dose prefilled disposable syringes..

Plastic Containers: 300 mg. in 50 ml. of 0.9% Sodium Chloride in single-dose plastic containers, in packages of 4 units. No preservative has been added.

ADD-Vantage®* Vials: 300 mg./2 ml. in single-dose ADD-Vantage® Vials, in packages of 25.

Exposure of the premixed product to excessive heat should be avoided. It is recommended the product be stored at controlled room temperature. Brief exposure up to 40°C does not adversely affect the premixed product.

'Tagamet' HCl (brand of cimetidine hydrochloride) Injection premixed in single-dose plastic containers is manufactured for SK&F Lab Co. by Travenol Laboratories, Inc., Deerfield, IL 60015.

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Glimpses of Alaskan Medical History

Edited by Robert Fortuine, M.D.

A Desperate Winter in Prince William Sound (1786–87)

During the summer of 1786 the English Captain John Meares brought his ship *Nootka* to the Gulf of Alaska to investigate trade possibilities. After a brief stop in Cook Inlet, he settled down to trading with the Chugach in Prince William Sound, where the lateness of the navigation season caused him to overwinter near the present village of Tatitlek. Initially the crew members remained in good spirits and local food was plentiful. Soon, however, the waterfowl migrated south, the ponds froze over, and the sound became too stormy to hunt or fish. The local Natives, who had earlier supplied the ship with food, departed to their winter villages, where they faced their own problems of subsistence.

After a terrible winter the survivors of the *Nootka* were finally rescued in mid-May by the crew of another English ship, under the command of George Dixon. Conditions were already improving by this time because of the availability of fresh food. Scurvy had taken a grim toll, however; 23 of the original 45 men had perished.

The text presented is taken from Meares's account of the voyage (1). Several interesting features of scurvy should be noted. It was widely believed in the 18th century that scurvy was caused, at least in part, by poor ventilation in crowded ships' quarters. Clinically, depression ("melancholy") was often a prominent symptom — a black state of mind which seemed to drain all energy and dampen the will to live. It is apparent from the text that the "pine" (probably spruce) needle remedy, although unpleasant, was effective, yet few could be persuaded to exert themselves to take it. Finally, exercise was thought to be a major method of treatment in this period. One can clearly picture a company of wretched men racked with pain dutifully performing calisthenics in the damp cold air.

"For some time we kept in the fires night and day, but the smoke which proceeded from a temporary stove, made out of one of the forges, was so troublesome, that the people, who were now falling ill, were fully convinced that this continual smoke was the cause of their sickness. After the heavy fall of snow we had twelve down with the scurvy, and towards the end of the month four died, and the number encreased to twenty-three who were confined to their beds, amongst whom was the surgeon, who was extremely ill. The first officer on finding himself slightly affected in the breast, a symptom which generally foreboded a fatal determination in a very few days, got rid of it by continually chewing the young pine branches, and swallowing the juice; but, from the unpleasant taste of this medicine, few of the sick could be prevailed upon to persist in taking it.

"At the latter end of February the disorder had encreased, and no less than thirty of our people were so ill that none of them had sufficient strength to get out of their hammocks: — four of them died in the course of the month. — Indeed, at this time, our necessaries were so far exhausted, that if the more violent symptoms of the disorder had abated, there was a want of proper food to complete the cure. These melancholy circumstances were rendered more afflicting by the hopeless minds of the crew; for such was the general discouragement amongst them, that they considered the slightest symptom of the disorder to be a certain prelude to death....

"The month of March brought no alleviation of our distresses: — It was as cold as the months that preceded it. In the early part of it there fell a great deal of snow, which encreased the number of the sick, and the violence of the disorder in those who were already afflicted by it. — In the course of this month we had the melancholy office of performing the last imperfect obsequies to the remains of the surgeon and the pilot. These were heavy misfortunes, and the loss of the former, at a moment when medical knowledge was so necessary, must be considered by all who read this page, as a consummate affliction.

"The first officer finding a return of his complaint, applied to the same means of relief which had before been so successful, — exercise and the juice of the pine tree. — He made a decoction of the latter which was extremely nauseous, and very difficult, though very much diluted, to keep on the stomach: — it operated repeatedly as an emetic, before it became a progressive remedy: — and perhaps this very effect, by cleansing the stomach, aided the future salutary operations of this anti-scorbutic medicine. The second officer and one or two of the seamen persisting in the same regimen, found similar benefit, and were recovered from a very reduced state; but it is one of the unfortunate symptoms of this melancholy disorder, to be averse to motion, and to find pain bordering on anguish, in attempting to use that exercise which is the predominant remedy."

REFERENCE

1. Meares, John. Voyages made in the years 1788 and 1789 from China to the northwest coast of America. London: Logographic Press 1790. Pp xvii–xx.

News from the American Society for Circumpolar Health

Summary of Hepatitis B Workshop
7th International Congress on Circumpolar Health
Umea, Sweden
June 12, 1987

by Brian J. McMahon, M.D.[1]

A workshop on hepatitis B in circumpolar countries, attended by more than 50 people, was held on Friday, June 12, 1987 in Umea, Sweden. During the first hour of the workshop, papers on the epidemiology of hepatitis B in North America were presented.

Dr. Minuk and colleagues from Calgary presented data suggesting that chronic hepatitis B surface antigen (HBsAg)-positive Inuit carriers are more likely to have an Epstein-Barr virus (EBV) serological profile consistent with a chronic EBV infection than matched HBsAg-negative controls.

Dr. Larke and associates from Edmonton reported the results of an extensive serological study of hepatitis B infection that included almost all of the communities in the Northwest Territories in Canada. Of 14,198 persons screened of Inuit, Dene, and non-Native ethnicity, 3% were positive for HBsAg and 20.7% were positive for anti-HBs. Hepatitis B "e" antigen (HBeAg) was detected in only 8.8% of HBsAg-positive persons.

Dr. Baikie and colleagues presented data on the epidemiology of hepatitis B virus infection in northern Labrador, Canada. Of 1,505 persons screened in five communities, HBsAg was found in 3.9% and evidence of HBV seromarkers in 16.7%, with rates of HBsAg found in 7.1% of the Inuit population. No HBsAg carriers were HBeAg-positive and seropositivity was only 3.8% among those below the age of 20.

Dr. Robinson and colleagues reported on hepatitis B prevalence among the James Bay Cree Indians in Quebec, Canada. Over 1,600 individuals from six villages were screened in two serosurveys. In one high prevalence village, HBsAg was found to be present in 3.4% and anti-HBs in 18.9%. Prevalence of HBV markers in this village increased with age until age 40-44, and 12.2% of individuals under 14 years of age were found to have positive markers. Seromarker prevalence was 6% in the five other villages together.

Drs. Tower and Middaugh presented the prevalence of hepatitis B infection in the non-Native population of Alaska. High prevalence rates of HBV were found in Asians, but low prevalence rates were found in Caucasians who belonged to recognized low-risk groups. High prevalence rates of HBsAg (10%) were found in male homosexuals and intravenous drug users.

Dr. Wainwright and colleagues presented data on the long-term immunogenicity and efficacy of HBV Vaccine in Yupik Eskimos. Persistent levels of anti-HBs were found five years after the initiation of hepatitis B vaccination in 81% of Eskimos who were shown to respond to the vaccine. In addition, breakthrough infections were found in only three of the responders, and none of these persons had symptomatic hepatitis or were HBsAg-positive.

During the second hour of the conference, papers on the control of hepatitis B as well as other papers were presented. Dr. McMahon and colleagues presented a paper on the comprehensive program for the primary and secondary prevention of hepatitis B virus infection and primary hepatocellular carcinoma in Alaskan Natives. In Alaska, over 55,000 Alaskan Natives have been tested for HBV seromarkers and 3% were found to be HBsAg-positive, with 18% found to have anti-HBc. Hepatitis B vaccination series has been initiated or completed in 37,000 individuals. In the area with the highest infection rates, the incidence of acute symptomatic HBV has dramatically dropped from 215/100,000 to 14/100,000 after completion of immunization of 90% of the serosusceptibles. As a result of a program to screen all HBsAg-positive persons with alpha-fetoprotein (AFP) twice yearly (presented in the cancer session on June 11), the one-year, case-fatality rate from hepatocellular carcinoma has decreased from 100% to 50% due to the diagnosis of small resectible lesions in seven patients.

Dr. Larke then presented the results of the immunization program in the Northwest Territories of Canada. Communities in the previously reported serosurvey found to be at increased risk of hepatitis B were offered immunization; 94% of the target population of 1,612 receiving the first dose of vaccine. This vaccination program is currently being extended to other Northwest Territories regions.

Dr. Magonet and his colleagues then presented a vaccination program in the Crees of northern Quebec. In the villages previously described by Dr. Robinson,

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[1] Chairman of the Hepatitis B Workshop held on June 12, 1987 at the 7th International Congress on Circumpolar Health held at Umea, Sweden.

90% of serosusceptibles had begun or completed their immunization series.

In the next half-hour, several participants from Scandinavian countries presented data on hepatitis B infection in their areas. Dr. Peter Skinhoj presented updated serosurvey data from Greenland Eskimos. Again, it was shown that there was high prevalence of HBsAg, approximately 10% in Greenland Eskimos, similar to what was reported in the mid-1970s. Antibody prevalence rates increase sharply in the 15- to 24- year age group to 50 percent, indicating an intense, sexual, spread of the infection within this age group. Other areas in Greenland, however, showed lower infection rates comparable to the Canadian figures. In the earlier cancer session, researchers from Greenland had also reported that despite the high prevalence of hepatitis B surface antigen, there was a low prevalence of primary hepatocellular carcinoma.

Dr. Erik Nordfeldt then presented data on hepatitis B and delta infection in Sweden. The number of acute hepatitis B cases in Sweden during the last four years have been stable, about 500 cases per year, giving an incidence of about 6/100,000 inhabitants. However, during the same period the reported number of carriers has risen from 165 to 453. This rise is explained by the high immigrant rate from Asia and other areas with a high carrier rate. Hepatitis delta infection was introduced in Sweden in the beginning of the seventies and has rapidly spread among addicts, reaching a prevalence of 80% of anti-delta among HBsAg-positive addicts in the beginning of the eighties.

Dr. Lars Magnius presented information about HBV DNA in HBsAg-positive carriers; only approximately 12% of those with anti-HBe were found to also be HBV-DNA positive. The only exceptions were in patients with renal transplants or on renal hemodialysis where there was a significantly higher prevalence of HBV-DNA in HBe-negative carriers.

Finally, Dr. Lars Hambreaus presented data to show the effectiveness of reduced dosages of hepatitis B vaccine given intradermally in young adults.

Over the next half-hour there was a discussion on control of hepatitis B infection in circumpolar areas.

Dr. Minuk pointed out that in parts of Canada, such as Baker Lake area, transmission of hepatitis B appears to have stopped 20 years ago, since HBV sereomarkers are uncommon in persons under 20 years of age, and all HBsAg-positive carriers from this area are HBeAg and HBV-DNA negative.

Dr. Larke found the same phenomena in several of the villages that he surveyed in the Northwest Territories. However, in a small cluster of villages in the western part of the Northwest Territories and in several communities on Baffin Island, there was a relatively high prevalence of seromarkers among persons under 20 years of age; most of the individuals that were positive for HBeAg were young people living in these areas.

Dr. Robinson noted that a follow-up serosurvey of Cree Indians in northern Quebec showed that several seroconversions as well as some acute clinical cases had occurred.

While Alaska had an extensive control program among Alaskan Natives in place and immunization campaigns had been carried out in selected populations in northern Canada, other Circumpolar areas had plans for hepatitis B control programs. In the Northwest Territories of Canada, all pregnant women have been screened for HBsAg since 1981 and infants born to HBsAg-positive mothers are offered immunization with both HBIG and HB vaccine.

Dr. Jens Misfeldt mentioned that in Greenland, vaccination since January 1987 has been recommended for high-risk groups such as newborn infants of HBsAg-positive mothers, stable sexual partners of carriers, and health care personnel. Testing of pregnant women is compulsory and blood donors must be tested once a year. A prospective follow-up of carriers is being considered.

A discussion then proceeded to what other control measures could be undertaken in circumpolar areas. It was suggested that countries begin to plan more wide scale control programs since the price of hepatitis B vaccine should dramatically drop in the next few years as more pharmaceutical companies begin to market hepatitis B vaccines. Suggestions include consideration of immunizing all household contacts of HBsAg-positive persons and immunizing all newborns in communities where there is a high prevalence of HBV seromarkers in young adults and children, communities where there is evidence of acute clinical hepatitis B infections or asymptomatic seroconversions, and in communities which have a high prevalence of HBeAg-positive carriers. When the price of hepatitis B vaccine drops to a reasonably reduced level, circumpolar countries would then consider, in addition to the above measures, immunization programs aimed at all children and sexually active adults. Eventually, the price of the vaccine may be reduced to an extent where all seronegative persons could be offered immunization.

Discussion then ensued on the follow-up of HBsAg-positive carriers. Several individuals felt that it was important to follow all HBsAg carriers for several reasons. First of all, the incidence of primary hepatocellular carcinoma is not known in circumpolar areas of Canada where HBV infection is prevalent. Secondly, in order to confirm previous observations that the incidence of hepatocellular carcinoma is low in Greenland Eskimos, a large number of HBsAg-positive Greenland Eskimos should be followed for the development of hepatocellular carcinoma to determine if there indeed is a reduced risk of development of HCC in HBsAg-positive carriers of Greenland Eskimo descent. If this is the case, then a search for the absence of co-factors which cause hepatocellular carcinoma or the presence of protective factors in Greenland Eskimos should be initiated. Finally, since alpha-fetoprotein screening has been shown in Alaska and other areas to detect the majority of HCC cases at a resectible and potentially curable stage, HBsAg-positive carriers in circumpolar countries could be followed with AFP screening every 6-12 months to detect HCC at a resectible stage. Establishment of parallel studies following HBsAg-positive carriers conducted in respective Circumpolar countries with high prevalence of HBV infection would be of great scientific as well as public health importance. Differences in the incidence of HCC could be determined and the usefulness of alpha-fetoprotein or other tumor markers in screening for HCC could be further delineated.

The hepatitis B workshop stimulated a great deal of interest and enthusiasm from the participants. It was felt that this workshop should again be held in three years at the VIII International Conference on Circumpolar Health to be held in Whitehorse, Canada. It was suggested that Canadian scientists or public health workers host this conference. In the meantime, it was also suggested that an interim meeting on hepatitis B in circumpolar areas be held in Canada in approximately 18 months.

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This workshop was sponsored by the American Society for Circumpolar Health with a generous grant from Merck, Sharp, & Dohme Company.

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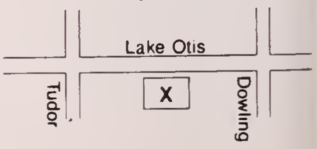

News from the American Society for Circumpolar Health

Albrecht Honored as Circumpolar Health Medical Pioneer

Zoonoses, virus diseases and alcoholism are uniting countries at the top of the world.

These health problems, and many others inherent in the northern latitudes, cross boundaries with no concern for nationalities and invade lives with relative ease. So, medical professionals from lands surrounding the North Pole created the International Union for Circumpolar Health to address these health issues and share information and concerns.

Dr. C. Earl Albrecht, founder and first president of the 20-year-old Circumpolar Health Symposium which became the Circumpolar Health Union, was honored earlier this year with one of the first Hildes medals to be awarded by the organization. The large golden medallion was presented for "humanitarian services to the people of the North and outstanding contribution to the integration, coordination, and promotion of circumpolar health and research services in the Arctic and Subarctic."

The slight, 82-year-old medical doctor who originally came to Alaska in 1935 to be a medical missionary has been a part of the "union" since it's inception in 1967.

Albrecht referred to his work in the union as an opportunity. "I was in a position to fulfill a dream," he said, "to develop the circumpolar health movement, which I call it." The former Matanuska Valley doctor developed a concern for the unique problems of the arctic while treating the inhabitants of Alaska, serving as Alaska Territorial Commissioner of Public Health, and meeting with medical people in nearby polar areas.

"All of us were concerned about similar problems," Albrecht said of his meetings with those doctors.

So with a $65,000 grant from the United States Public Health Service, Albrecht, who was then a governor of the Arctic Institute of North America, organized the first symposium. Speakers and guests at that first exchange represented Canada, Denmark, Greenland, Finland, Norway, Sweden and the USSR, in addition to the United States.

"I think the importance of the organization was that it proved to be worthwhile, ... so much so that Europeans said 'We must have the next meeting'."

Photo courtesy of Alaska and Polar Regions Department, Elmer E. Rasmuson Library, University of Alaska-Fairbanks, Alaska Nurses Association Collection

The Circumpolar Health Union is different from most medical and scientific organizations, Albrecht said. "Doctors like myself attend our own medical and public health association meetings, but this is different. We have a bond because of permafrost, cold weather and dark nights. We have common problems like alcoholism and psychiatric problems and viral and enteric diseases that remain with us, and there is emphasis now on pollution."

Although topics of emphasis vary at each symposium, some concerns related to living in the north have remained unchanged in the past 20 years. That first conference featured papers on pulmonary and viral diseases, zoonoses (diseases that can be transmitted to man by vertebrate animals), environmental stresses on human behavior, physiology, anthropology, nutrition and hazardous contamination of the environment.

As for the future, Albrecht, who still spends his summers in Alaska, emphasized the importance of research involving the aboriginal people, particularly in areas of rapid development. "Communities can become laboratories while searching for information on accidents, suicide, homicide and alcoholism," he said.

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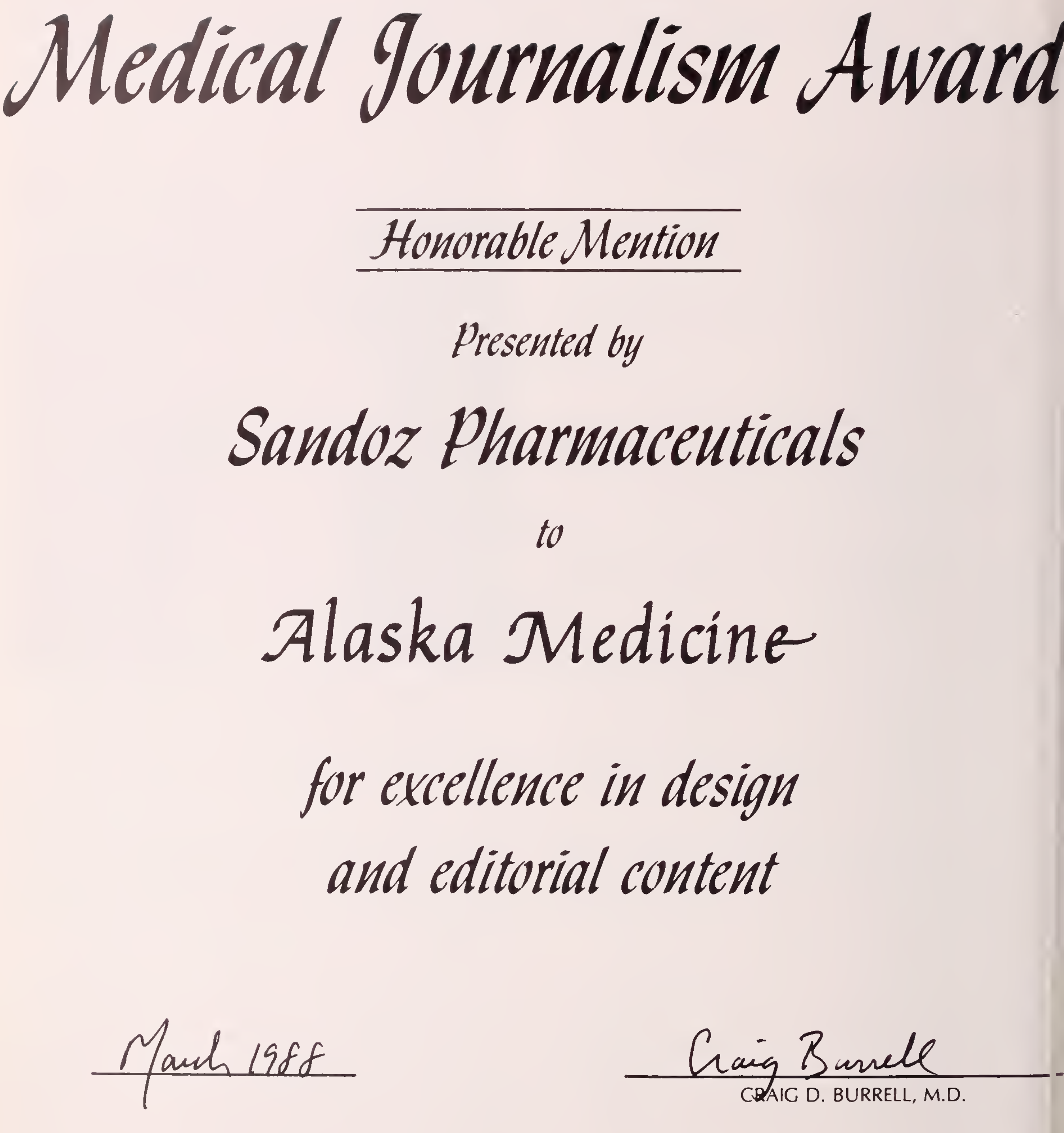

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March 1988

Craig Burrell
CRAIG D. BURRELL, M.D.

My Turn

Veterans' Administration Plans Changes—Can Alaska's Health Care System Survive?

Currently, because there are no VA hospitals in Alaska, veterans in Alaska are seen, cared for, and treated in their own communities by their own private practice physicians in their own local hospitals. Our system provides very high quality care, and patients express a high level of satisfaction with the system. Formidable problems of delivering health care to our widely dispersed population have largely been overcome. The volume of health services provided to Alaska's veterans accounts for approximately 10–15% of the total patient services provided by hospitals across our state.

A considerable amount of money has been spent in these communities in expanding and renovating facilities, and in developing and providing new services and new technologies so that all Alaskans can enjoy better health. For example, the state recently spent $10 million building a new hospital in Cordova—and additional expenditures are being planned for other communities to upgrade their facilities. Humana Hospital and Providence Hospital recently completed major expansion projects, adding new technologies and new services. All of us have benefitted.

Alaskans now do not have to leave our state to obtain sophisticated, expert, state-of-the-art health care like we did as recently as 15 years ago, because these services using the latest technologies and employing the highest degree of expertise are now available.

But, the availability to all Alaskans of this high level of care is threatened by proposed VA plans to restrict options for health care of Alaska's veterans. The VA wants to add 55 inpatient hospital beds to a new military hospital that is planned to be constructed at Elmendorf Air Force Base. The VA plans to treat all Alaskan veterans there, taking these patients away from their local physicians, communities, and hospitals. The VA is well along with its plans.

Why do this? The VA says it will save money. Most others do not share this opinion. But, even if some savings were to accrue to the VA, what would be the impact of the VA plans on the rest of Alaska? The VA has not looked. But, others have.

The loss of business that would result from removing all veteran health care from Alaska's private sector would severely impact Alaska's already fragile health care delivery system. Because the money has already been spent to add new technologies, services, and facilities, costs can not simply be reduced if veterans' services are eliminated from the private sector. And veterans' health care services accounted for 10–15% of the patient load in Alaska.

Not only will the VA proposal affect the veterans by requiring them to go to Elmendorf Air Force Base hospital but it also will affect the private sector by increasing their costs, possibly resulting in less access to health care for all Alaskans. Since the monies have already been spent in renovating and expanding, these fixed costs will have to be passed on to remaining payors. The result—higher costs to them. This will significantly increase the cost to the State of Alaska due to Medicaid and to state employees because health insurance costs will increase significantly.

The VA argues that they will save money by piggybacking onto the military hospital. However, several independent studies have cast grave doubts about the reliability of these optimistic projections. The cost of health care in Alaska is already of concern—this proposed action by the VA will only make the situation worse. In fact, should the VA proceed with their current plans, it is likely that VA costs will increase and costs of health services to all other Alaskans will increase as well.

One of the inescapable problems that we face is that we have a very small population. While the latest technologies and services are now available so that we no longer have to leave the state for sophisticated health care, the costs of providing state-of-the-art technology can only be spread over a very small number of people. In the lower 48, the cost of this same technology is shared by a vastly greater number of people.

Alaskans involved in the delivery of health care services—hospitals, nursing homes, physicians, dentists, pharmacies, and many others—make up the second largest group of employees in Alaska, second only to the state itself. We will all be severely impacted if the VA proceeds with its plans.

Up to this time, VA administrators and planners have failed to take into account the potential devastating impact of their proposed plans on all Alaskans. We and they need to look carefully at the whole picture instead of just one segment of it.

George Schneider, Chairman
Health Association of Alaska and
Alaska State Medical Association
Task Force

President's Page

Dear Alaskan Physicians,

I hope that this has been a good winter for you. I hope that your practice is busy and that you are enjoying the good life. After such a heady greeting I would like to get to the heart of a matter that has been bothering me since our council meeting in February in Juneau.

It seems that the council had its annual task of deciding who would be given the annual awards, such as "Physician of the Year" and the A.H. Robbins award for community service. Interestingly enough, there were several nominations from outside of ASMA for the annual community service award. Surprisingly, and maybe not so surprising, I should say we had a difficult time deciding upon the receipient of that award for the very simple reason that most of the nominees were all doing great things, but they were all medically-related in a sense. Dr. Gary Hedges made a very impassioned plea that physicians need to become more involved in their communities in relms outside medicine, and he listed any number of things, such as becoming involved in the school board, PTA, your church, or local civic organizations such as Rotary, Kiwanis, Lions Club.

I feel sure that we have physicians in this state who are doing just those things and performing an exceptional job, but we're certainly not aware of them. I guess what I am asking is this. I would ask each of you to do this. Take a good look at your lifestyle as far as your practice is concerned, your family, your hospital efforts, etc. and then take the time—let me say make the time to become involved in community affairs. I think it's exceedingly important that physicians become more visible outside the arena of medicine. I think that this statewide effort would certainly improve the image of physicians. Maybe we can get away from the image of being money-hungry, power-mad physicians as we have been so labeled by certain other professions.

Again a reminder of your annual convention which is to be held in Fairbanks on June 8-11, 1988. ASMA is your voice in the political arena of medicine, it is your voice for organized medicine in this state. I urge you to come to the meeting — attend the council meetings and the House of Delegates sessions. Speak your voice, let your wishes and your concerns be known to the officers and councilors of ASMA. We are here to serve you. Please give us a chance to do so.

Respectfully,

Jerry Little
ASMA President

Auxiliary News

Alaska State Medical Association Auxiliary

Last night medicine in Alaska scored a resounding victory with the election of several doctors and spouses to important precinct officer and convention delegate slots. The Republican caucuses were held in Anchorage with strong medical participation. For many medical people, this was a first experience in politics and what is significant is the enthusiasm of the participants. I heard numerous times, "I didn't know it would be so much fun." Two precincts adopted pro tort reform platforms.

The newly elected delegates will attend the district conventions in March where elections will take place for the state convention. At the district convention significant work will be done on platforms and resolutions. At the state level more elections will take place with the winners going to New Orleans for the national convention.

Medicine no longer has the luxury of leaving the business of politics to lawyers and business people. We are the second largest employer group in the State of Alaska and we deserve to be heard. Congratulations to our delegates.

Cassie Hill
President, ASMA Auxiliary

Executive Privilege

David Johnson, M.D., and I have just returned from our annual trek to Washington, D.C., where we met with our Alaskan Congressional delegation. This year I was privileged to represent three groups in our discussions as: executive director of the Alaska State Medical Association, chairman of the Health Care Coalition of Alaska (HCC), and as a board member of the Veterans Administration/Department of Defense (VA/DOD) Task Force. It is in this latter role that I will attempt to present a broad overview of the veterans administration's attempt to construct a new hospital, and failing in that attempt, to dovetail with the Air Force in renovating or constructing a new Elmendorf hospital to house veterans.

I suspect most of you are familiar with the Veterans Administration's release of the Inspector General's Audit and their public pronouncement that medical care payments in Alaska ballooned from $17.2 million in 1982 to a staggering $45.9 million in 1985. The Alaska State Medical Association responded in June, 1986 with the release of a detailed analysis of the VA report that outlined serious flaws in the manner in which the information was gathered and questioned the authenticity of the numbers, themselves.

In response to our analysis, the Veterans Administration, Alaska State Medical Association, and Health Association of Alaska began a series of meetings that spanned a twenty-month period and which resulted in the christening of a VA outpatient facility in Anchorage. These negotiations were particularly difficult in that the VA sent new players to each meeting with a slightly different message. But, finally an agreement was concluded with General Thomas Turnage, Administrator of Veterans Affairs. The General assured us that the VA's primary concern was to cut costs and that any movement towards development of a VA inpatient facility was in the far distant future.

But, even as we were being reassured by General Turnage, the Army Corps of Engineers were gathering data which would be released as the "Economic Analysis for Sharing Federal Medical Resources." This study would be the catalyst for involving the Air Force in a joint project to create at least 15 beds for the VA's needs. This scheme was designed to elicit support from veterans and the private sector, knowing of our sympathies towards the Air Force and their need for a new facility.

It was at this point that the Alaska State Medical Association and the Health Association of Alaska created a task force that could study, coordinate and respond as a single voice. In order to respond effectively, we needed the data and information from which the VA made their initial statement and their judgment that health costs in Alaska were out-of-control. We have requested these data many times over the past twenty months so that we could make responsible assessments, BUT AS OF THIS DATE, NONE HAS BEEN FORTHCOMING.

We then decided to employ the prestigious national firm, Arthur Anderson & Co., to study the health care system in Alaska as it relates to the Veterans Administration. Judgments then can be made in the best interest of Alaskan veterans. This study was completed and released in the middle of March 1988. However, when we employed Arthur Anderson & Co., we agreed to their stipulation that bits or pieces of the study not be released, even by those with the best intentions. We agreed that the study must be released as a whole document. But, I am permitted to state that the health care system in Alaska is a fragile entity and the quality of care issue does not appear to have been considered adequately in the VA's proposal.

I was instructed by the VA/DOD Task Force to deliver these studies to our congressional delegation and to provide additional information as needed. Senator Frank Murkowski, the ranking minority member of the Veteran's Affairs committee has been a staunch and able supporter of our concerns, and his staff has been intimately involved reviewing all the data for the Senator.

Upon review of the facts, Senator Murkowski has continuously opposed any additional VA beds in Alaska. The Senator has assured us that the Veterans Administration will not expand beyond the current level of care in Alaska.

Senator Ted Stevens, as chairman of the Defense Appropriations Committee also assured us that, apart from our concerns, the state of the federal budget prohibits any VA expansion in the foreseeable future. However, if the cost of care does not shrink dramatically, we should expect the VA to seek care in the lower 48 through the use of military transportation. We expressed additional concerns, however, that the VA could set aside funds now to do an engineering and site study. Senator Stevens assured us in a most positive manner that this would and could not occur.

Congressman Don Young has been apprised of our position and activities on this issue. He is supportive and offered any assistance he could provide.

Dr. John A. Gronvall, Chief Medical Director of the Veterans Administration, will be entertained by the Task Force during the month of April. We hope that Dr. Gronvall will study the Anderson report. We are confident that he will, after serious thought and careful study of all the facts, seek a solution that is in the best interest of Alaskan veterans. After all, the quality of care is the single most important issue! Our veterans deserve the best.

Editorial

Public Health Policy in Alaska—Who Needs it?

The health of Alaskans has never been better! The past three decades have seen an almost miraculous reduction in life-threatening infections and communicable diseases. Alaska's infant mortality rate has dropped to an all-time low—8.3/1000 live births. The life expectancy of an Alaskan born in 1988 has never been higher—75 years. Death rates also have fallen to an all-time low—from 980/100,000 in 1950 to 385/100,000 in 1985.

So why do we need to worry about public health policy? Why should we get excited about another call for action? If all these good things are true, how come all my friends are constantly complaining of symptoms of illness? And why are health care costs so high?

The State of Alaska has an extraordinary record in its past commitment to public health. Catastrophic epidemics of tuberculosis and other infectious diseases that ravaged Alaskans in the early 1950s have been largely eliminated by public health programs—implemented through an unusually effective partnership of local communities, Indian Health Service, and State of Alaska. Our not-so-recent past provides many examples of the effectiveness of public health programs in controlling diseases and preventing suffering, disability and death.

But we live in a dynamic and everchanging world. We must work constantly to maintain the painstaking successes that have been achieved. Public health efforts have been likened to "clearing the jungle." Without sustained efforts, the jungle regrows.

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We must also expand our activities to areas where success can be assured if we apply our available knowledge. And we must always be ready to respond to new health threats such as AIDS.

Public health—defined by many as social justice—is an absolute responsibility of effective government. The role of government must be as a "residual guarantor" responsible for assuring the provision of preventive services to all. To assure that benefits accrue to our entire population, we need a clearly defined public health policy. We need to reaffirm the commitment of the government of the State of Alaska to public health. In order to do so, we must define and share this commitment at the local, state, and federal level. Only then can it be that the state has reaffirmed its role as "residual guarantor." Only then can we achieve social justice.

Where services are readily available, government may be involved in delivering them. Where gaps exist, however, it is the responsibility of government to fill them. In many cases, however, governmental responsibilities overlap and, in these cases, there needs to be clear agreement on the division of responsibility. Finally, government must monitor the effectiveness of the efforts to control health problems. Crucial is the need for the state to assume a leadership role.

Public health confronts many complicated problems and is beset by many seeming paradoxes. Why, for instance, if we're so healthy, do we feel so sick? One reason for this seeming paradox is that we don't realize how much healthier we have become. Alaska is the nation's youngest state; 90% of all Alaskans are less than 50 years of age. One consequence of being young is that our memories are short. Few of us can recall just how bad was the health of Alaskans only a short time ago. In just 30 years we have eradicated many killers—smallpox, diphtheria, tetanus, tuberculosis, measles; we have dramatically reduced deaths in childbirth; and deaths from stroke have all but disappeared.

Recently, I spent another week (my fourth during the past two years) in Juneau reading death certificates. My time was primarily spent studying each certificate and looking up appropriate codes from a book, the International Classification of Diseases, to assign the right code to the cause of death and to other medical conditions recorded on the certificate. I read more than 10,000 death certificates from 1950 and 1980–86.

The number of deaths in Alaska in 1950 from infectious diseases, especially tuberculosis, is mind boggling. I had constantly to search the book to find the correct codes for an array of infectious diseases that I had not discovered among all the deaths I had coded for the years 1980-86. Sadly, I now know by heart the codes for rheumatic fever, rheumatic heart disease, polio, measles, pertussis, absesses, sepsis, gangrene, tuberculosis and its complications, diarrhea, peritonitis, appendicitis, and maternal death during childbirth. In 1950, many newborn infants died with a diagnosis of "too small to live." Infant mortality was shockingly high not only from tuberculosis and measles but also from malnutrition, failure to eat, neglect, starvation, and the cold. Many deaths in 1950 were caused by intracerebral hemorrhage (stroke) and uncontrolled hypertension (high blood pressure). There were only a few such deaths reported in Alaska for 1980-86. The improvements in our health are so great as to be difficult to comprehend.

Why are we doing better but feeling worse? As I said, one reason is that we have short memories so we don't realize how much healthier we have become. A second is that we have eliminated many diseases and conditions that caused early death. As our death rates dropped, our life expectancy increased. Thus, more people are living longer. But, because we are living longer, we are increasingly subjected to chronic diseases and degenerative disorders. A third reason is that we have developed high expectations for health. For example, we see our top athletes incur devastating injuries only to return to ever more impressive accomplishments. If they can do it, why can't we? Our expectations of the power of medical technology are misplaced. Few ever are aware of the tremendous, permanent disability experienced by these athletes in their later years. Fourth, there has been widespread commercialization of health. It's hard to sell health products to the healthy. We are bombarded with commercials and media messages telling us we are suffering from illnesses that the seller's remedies will cure. Finally, we have become increasingly aware of and concerned about changes in our environment that might affect our health. How does this relate to the need for a public health policy?

We must continue public health preventive programs for which we have absolute evidence of their effectiveness. Our efforts to maintain hard won successes can not be diminished. And, our greatest successes have been in prevention. Once illness strikes, our ability to treat often is limited. Progress must be measured in terms of reductions of preventable health problems—absence of illness, injuries and deaths—that based on previous experience might otherwise have been expected to occur.

New efforts are warranted in two areas. The first and most important is that we must use our available knowledge to "close the gap." What gap? The gap is the current burden of unnecessary illness that afflicts all of us. This burden is the illness that can be eliminated with knowledge that is already at hand. If we applied today the knowledge we have, two-thirds of all deaths would be postponed, and two-thirds of the potential years of life lost would be prevented.

We have seen miraculous improvements in the health status of Alaskans, but we can do better. We need to provide for social justice. We need to provide to all Alaskans preventive services based on current knowledge. Examples include preventing motor vehicle injuries by buckling up, preventing heart disease and cancer by eliminating cigarette smoking, reducing the tremendous burden of injury, disability and death from alcohol and drugs, improving safety and health at the worksite, reducing heart disease by cholesterol control, improved diet, and exercise, and avoiding treatable health problems by universal application of limited periodic health screening.

The second area where new efforts are warranted is responding to new epidemics such as AIDS and newly recognized health threats such as indoor air pollution. We must constantly seek and apply new knowledge through research and constant evaluation of our efforts.

We must demand that our government fulfills its role as our guarantor of good health. Prevention will save lives, will improve the quality of our lives and will, in the long run, save dollars. A clear public health policy developed by the State of Alaska will determine whether we pay for a fence at the top of the cliff or pay for ambulances and hospitals and rehabilitative services at the bottom.

A viable public health policy 1) will reaffirm our commitment of the role of government as our guarantor of health, 2) will enable us to determine our priorities, 3) will help us to allocate our resources and determine responsibilities for delivering public health services, and 4) will provide for monitoring the effectiveness of our efforts to control our health problems.

Government cannot provide public health; only we Alaskans at the grassroots can do that. But our government has an absolute responsibility to help us to obtain the benefits of today's knowledge. We must have a framework. We must establish objectives, set attainment levels, and establish a realistic time table.

If we meet these new challenges, 30 years from now the next generation of Alaskans will be healthier. They will benefit from additional huge reductions in avoidable causes of illness, disability and death. Today, we enjoy better health because of the effective and visionary leadership that was shown in the 1950s to respond and apply available knowledge to control epidemics at that time. The opportunities and the challenges to do the same, in turn, are before us. We must meet them by renewing our commitment to social justice and public health. Alaskans need a public health policy.

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Letters to the Editor

Malpractice and Patient Alienation

Dear Editor,

A lot has been written recently about the harmful effects of the malpractice situation, but no one seems to have identified what to me is the most unfortunate consequence of all; patient alienation. I've encountered more and more patients over the last several years who are doctor shopping because they feel that their current physician is hostile or insensitive or perhaps even employed by the insurance company or the Worker's Compensation carrier. In my early years of practice, I don't think I encountered this attitude as frequently as I'm seeing it now. I suspect that's because physicians today tend to regard each and every patient at arm's length and with a coolness easily sensed by the patients. These patients surely must come to feel that the doctor is not acting as their advocate but as an agent for some other party.

This unfortunate development of patient alienation plays directly into the hands of the attorneys. A person is far more likely to initiate a suit against a stranger or a non-supportive party than against someone who he feels is acting as a friendly advisor and confidant. Thus a malpractice suit is easily initiated by an attorney leading to further suspicion and hostility. The only winners in this destructive game are the lawyers.

Sincerely,
Richard W. Garner, M.D.

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James H. Sammons, MD
Executive Vice President

American Medical Association 535 North Dearborn Street; Chicago, Illinois 60610

Letters to the Editor

Donor Directed Blood Donation

Dear Editor:

I was quite disappointed to see the establishment of a directed blood donation program here in Anchorage and in the state.

While I fully understand there's been considerable demand and pressure to develop such a program for the general public, I do not believe there is any medical and scientific basis for such a program and, therefore, I feel it should not be offered.

As I'm sure you're aware, donor directed donation does not offer additional safety compared to blood used in a voluntary random pool. This has been well studied and, therefore, we are creating a service that has no medical or scientific basis. It has long been my feeling as has been the general feeling of the medical community that there ought to be at least some scientific basis for medical programs offered to the public. I believe it is this goal that has separated us from the chelators, the chiropractors, and the various other alternative health care services offered in the community which serve the patient "demand" but have no scientific basis.

Again, understanding public pressure I do believe it would have been better to have aggressively pursued an educational program so that we are not creating an illusion on the part of the public that donor directed transfusion is anyway safer than voluntary pooled blood. I also feel that donor directed transfusion policy may have some negative features. These include:

1) Creation of the illusion on the part of the public that there is a difference in terms of safety between directed and random volunteer transfusion.

2) There may be a decrease in voluntary donation if our volunteer donors assume that patients will either donate their own blood or receive donor directed transfusion and, therefore, reduce a valuable resource pool.

3) We may inadvertently create, therefore, a two-tier blood system for donor directed blood for those who have donors and a random pool for those who don't with again the illusion on the part of the patients who have no directed donors that they may be receiving an inferior product.

4) There are likely to be an increase in ancillary charges and procedural difficulties in handling donor directed transfusions.

5) There may be an actual increase risk of contracting AIDS, hepatitis, etc., in the donor directed transfusion group. Generally these donors are "first time donors" and, therefore, have not had the opportunity to have repetitive screening tests (as have the majority of volunteer donors). Therefore, these first time donors may be more likely to be in the "window period" for either hepatitis, AIDS, or other infectious diseases.

6) Perhaps again, theoretically, there may be some increased risk for donor directed transfusion in that these donors may be less likely to admit to homosexual activity, drug use, etc. which may make them high risk donors.

7) Patient safety may be compromised if a patient delays emergent transfusion while waiting for "safer" directed blood.

Again, I empathize somewhat because of the intense pressure to create such a program. But, the bottom line is that I believe the medical community as well as the blood banking system owes it to the public to offer programs that have scientific merit. Had other organizations caved into the unrealistic demands of the AIDS fearing public, many individuals would have been fired from their jobs, children barred from school and other unfair discriminatory practices performed in the name of AIDS fear.

I would hope, therefore, that the donor directed program be reviewed critically on a frequent basis, while continuing with public education so that this unneeded, unnecessary and, therefore, unwarranted service can be deleted.

Steven B. Tucker, M.D.
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CARAFATE®
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BRIEF SUMMARY

CONTRAINDICATIONS
There are no known contraindications to the use of sucralfate.

PRECAUTIONS
Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that the simultaneous administration of CARAFATE with tetracycline, phenytoin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. The clinical significance of these animal studies is yet to be defined.

Carcinogenesis, Mutagenesis, Impairment of Fertility: No evidence of drug-related tumorigenicity was found in chronic oral toxicity studies of 24 months' duration conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies have not been conducted.

Pregnancy: Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted i human milk. Because many drugs are excreted in human milk, cautio should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not bee established.

ADVERSE REACTIONS
Adverse reactions to sucralfate in clinical trials were minor and only rarely le to discontinuation of the drug. In studies involving over 2,500 patients adverse effects were reported in 121 (4.7%). Constipation was the mos frequent complaint (2.2%). Other adverse effects, reported in no more tha one of every 350 patients, were diarrhea, nausea, gastric discomfort, indi gestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo

DOSAGE AND ADMINISTRATION
The recommended adult oral dosage for duodenal ulcer is 1 gm four times day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should no be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or tw treatment should be continued for 4 to 8 weeks unless healing has bee demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED
CARAFATE (sucralfate) 1-gm pink tablets are supplied in bottles of 100 an in Unit Dose Identification Paks of 100. The tablets are embossed wit MARION/1712.
Issued 3/8

References:
1. Grossman MI: *Scand J Gastroenterol* 58 (suppl 15):7-16, 1980.
2. Marks IN, in Hellemans J, Vantrappen G (eds): *Gastrointestinal Tract Dis orders in the Elderly.* Edinburgh, Churchill Livingstone, 70-81, 1984.
3. Krentz K, Jablonowski H, in Hellemans J, Vantrappen G (eds): *Gastrointes tinal Tract Disorders in the Elderly.* Edinburgh, Churchill Livingstone, 62-6 1984.

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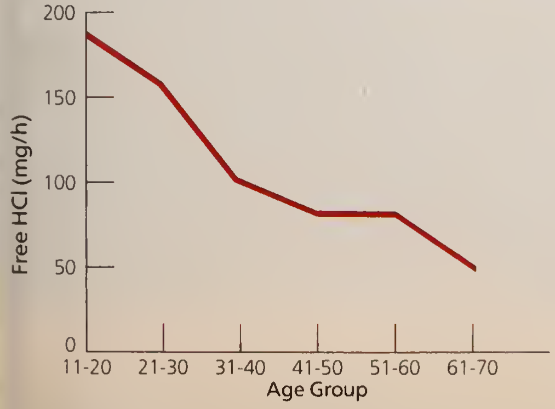

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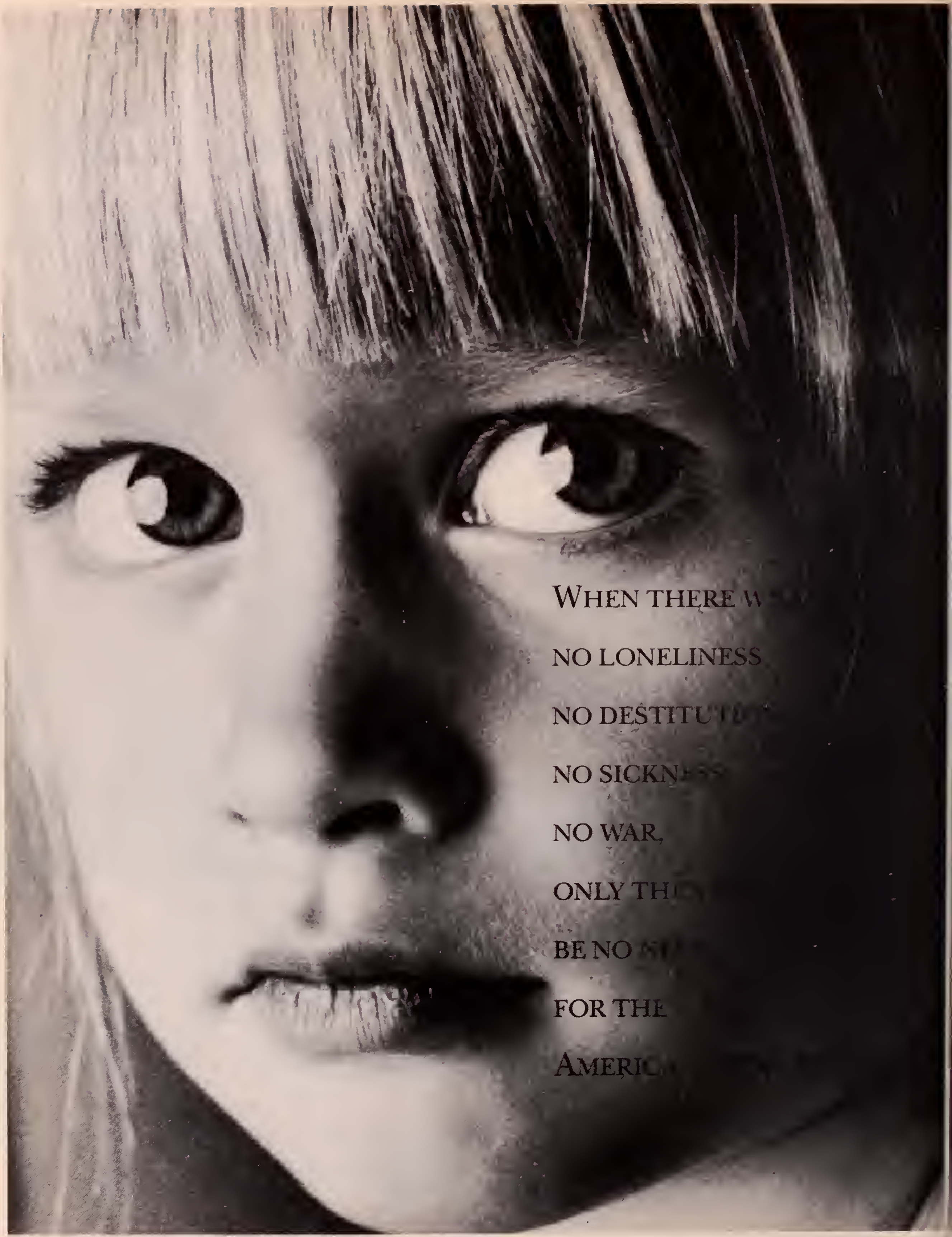

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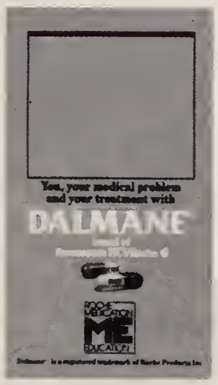
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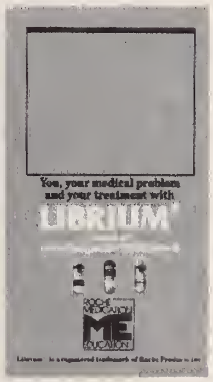
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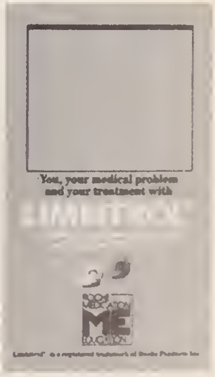
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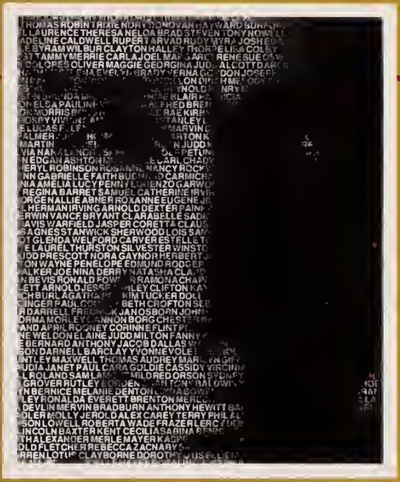

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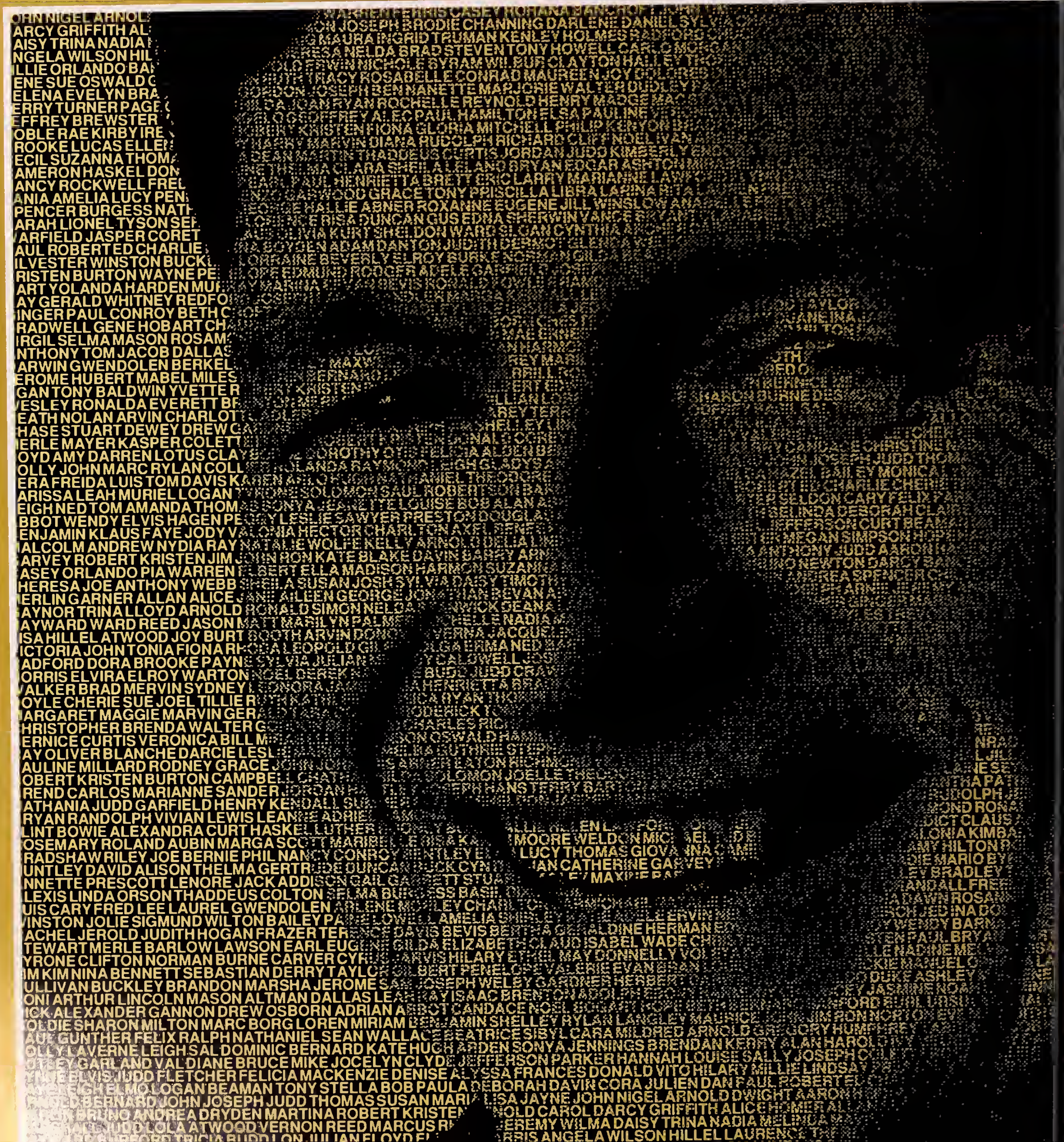

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...like the more than one million patients who have received INDERAL® LA.

In a recent survey, 4,120 participating physicians gave us their views[1] on INDERAL LA in the treatment of hypertension, angina and migraine.

INDERAL LA is their preferred beta blocker

...of the nearly three out of four physicians responding to the questionnaire, an impressive 97% rated INDERAL LA good to excellent for overall performance. Virtually all cited efficacy, tolerability, long-term cardiovascular protection and once-daily convenience as important factors in their choosing to prescribe INDERAL LA.

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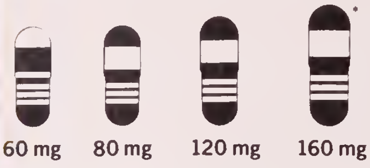

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

INDERAL® LA brand of propranolol hydrochloride **(Long Acting Capsules)**

DESCRIPTION. INDERAL LA is tormulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

CLINICAL PHARMACOLOGY. INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specitically competes with beta-adrenergic receptor-stimulating agents tor available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) tor the capsules are approximately 60% to 65% ot the AUCs for a comparable divided daily dose ot INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting trom the slower rate of absorption of propranolol. Over a twenty-tour (24) hour period, blood levels are tairly constant tor about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those ot two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end ot the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose ot conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses ot heart rate, systolic pressure, and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

INDICATIONS AND USAGE. Hypertension: INDERAL LA is indicated in the management ot hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

Angina Pectoris Due to Coronary Atherosclerosis: INDERAL LA is indicated tor the long-term management of patients with angina pectoris.

Migraine: INDERAL LA is indicated tor the prophylaxis ot common migraine headache. The efficacy ot propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

Hypertrophic Subaortic Stenosis: INDERAL LA is usetul in the management ot hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness ot propranolol hydrochloride in this disease appears to be due to a reduction ot the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

CONTRAINDICATIONS. INDERAL is contraindicated in 1) cardiogenic shock; 2) sinus bradycardia and greater than first-degree block; 3) bronchial asthma; 4) congestive heart failure (see WARNINGS) unless the tailure is secondary to a tachyarrhythmia treatable with INDERAL.

WARNINGS. CARDIAC FAILURE: Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, it necessary, they can be used with close tollow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Theretore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

IN PATIENTS WITH ANGINA PECTORIS, there have been reports ot exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate tor the management ot unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema) — PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation ot beta receptors.

MAJOR SURGERY: The necessity or desirability ot withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor ot beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA: Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manitestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS: Beta blockade may mask certain clinical signs ot hyperthyroidism. Therefore, abrupt withdrawal ot propranolol may be followed by an exacerbation ot symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid tunction tests, increasing T_4 and reverse T_3, and decreasing T_3.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case this resulted after an initial dose of 5 mg propranolol.

PRECAUTIONS. GENERAL: Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction ot intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS: Elevated blood urea levels in patients with severe heart disease elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS: Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL (propranolol HCl) is administered. The addec catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium channel-blocking drug, especially intravenous verapamil, tor both agents may depress myocardia contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of beta blocker and verapamil has resulted in serious adverse reactions, especially in patients witl severe cardiomyopathy, congestive heart failure, or recent myocardial intarction.

Aluminum hydroxide gel greatly reduces intestinal absorption of propranolol.

Ethanol slows the rate of absorption of propranolol.

Phenytoin, *phenobarbitone*, and *rifampin* accelerate propranolol clearance.

Chlorpromazine, when used concomitantly with propranolol, results in increased plasma levels c both drugs.

Antipyrine and *lidocaine* have reduced clearance when used concomitantly with propranolol.

Thyroxine may result in a lower than expected T_3 concentration when used concomitantly wit propranolol.

Cimetidine decreases the hepatic metabolism ot propranolol, delaying elimination and increasin blood levels.

Theophylline clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY: Long-term studies in animal have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in bot rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any ot the dosage levels. Reproductive studies in animals did not show any impairment ot tertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in anim studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be use during pregnancy only if the potential benefit justifies the potential risk to the tetus.

NURSING MOTHERS: INDERAL is excreted in human milk. Caution should be exercised whe INDERAL is administered to a nursing woman.

PEDIATRIC USE: Satety and effectiveness in children have not been established.

ADVERSE REACTIONS. Most adverse effects have been mild and transient and have rare required the withdrawal ot therapy.

Cardiovascular: Bradycardia; congestive heart failure; intensitication ot AV block; hypotensio paresthesia ot hands; thrombocytopenic purpura; arterial insufficiency, usually of the Raynaud typ

Central Nervous System: Light-headedness; mental depression manifested by insomnia, lassitud weakness, fatigue; reversible mental depression progressing to catatonia; visual disturbances; hallucinations; vivid dreams; an acute reversible syndrome characterized by disorientation for time ai place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased perfc mance on neuropsychometrics. For immediate tormulations, fatigue, lethargy, and vivid drean appear dose related.

Gastrointestinal: Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constip tion, mesenteric arterial thrombosis, ischemic colitis.

Allergic: Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching ai sore throat, laryngospasm and respiratory distress.

Respiratory: Bronchospasm.

Hematologic: Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

Auto-Immune: In extremely rare instances, systemic lupus erythematosus has been reported.

Miscellaneous: Alopecia, LE-like reactions, psoriasitorm rashes, dry eyes, male impotence, a Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the sk serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associat with propranolol.

DOSAGE AND ADMINISTRATION. INDERAL LA provides propranolol hydrochloride ii sustained-release capsule for administration once daily. If patients are switched trom INDER Tablets to INDERAL LA Capsules, care should be taken to assure that the desired therapeutic effec maintained. INDERAL LA should not be considered a simple mg-for-mg substitute for INDER INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessa especially to maintain effectiveness at the end ot the 24-hour dosing interval.

HYPERTENSION — *Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg or daily or higher until adequate blood pressure control is achieved. The usual maintenance dosag 120 to 160 mg once daily. In some instances a dosage ot 640 mg may be required. The time needed full hypertensive response to a given dosage is variable and may range trom a few days to seve weeks.

ANGINA PECTORIS — *Dosage must be individualized.* Starting with 80 mg INDERAL LA once da dosage should be gradually increased at three- to seven-day intervals until optimal response obtained. Although individual patients may respond at any dosage level, the average optimal dos appears to be 160 mg once daily. In angina pectoris, the value and satety ot dosage exceeding 320 per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (WARNINGS).

MIGRAINE — *Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once da The usual effective dose range is 160-240 mg once daily. The dosage may be increased graduall achieve optimal migraine prophylaxis. If a satistactory response is not obtained within four to weeks after reaching the maximal dose, INDERAL LA therapy should be discontinued. It may advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS — 80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE — At this time the data on the use of the drug in this age group are too limite permit adequate directions for use.

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Reference:

1. Data on file, Ayerst Laboratories.

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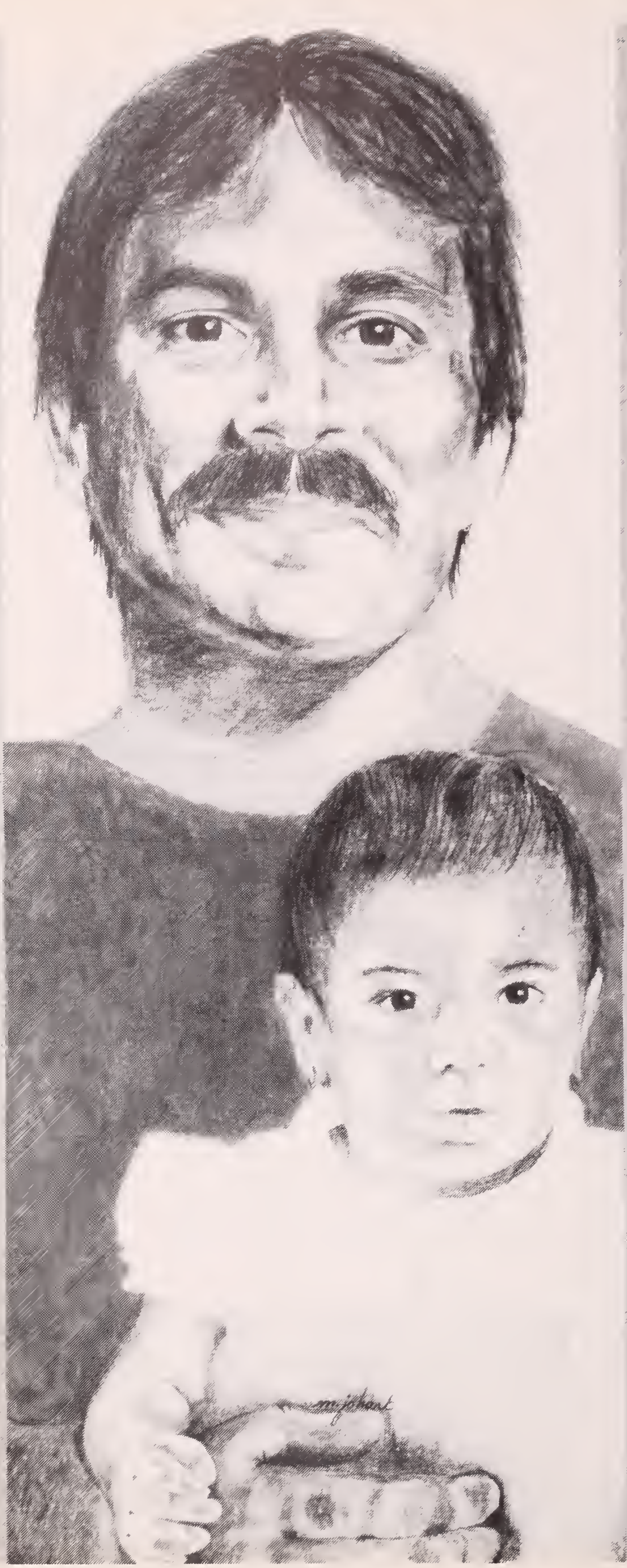

"For the poor you have always with you . . ."

John 12:8

The Bible tells us there will always be poor people. But nowhere do the Scriptures say that the poor should not receive treatment when they are sick or injured. Indeed, Christ admonished his disciples to "cure the sick and heal the leprous."

Today, leprosy is pretty much a thing of the past, at least in America. But poverty and illness are still with us. And Christ's call is still a challenge to us.

As a society, we have a responsibility to meet the needs of those less fortunate than ourselves. As a nation, we must find a way to provide necessary health care to everyone—rich and poor; young and old; male and female; yellow, red, black, and white.

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Carbon Monoxide Exposure in Mountaineers on Denali

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ABSTRACT

Carbon monoxide (CO) poisoning is a particular hazard for persons camping in cold, stormy, high altitude locations such as Mt. McKinley (Denali). Recently, two climbers were killed by CO on Denali while cooking in a tightly sealed tent. CO has also contributed to high altitude illness. To assess exposure in various shelters we measured CO levels with a portable monitor in a mountaineering tent, igloos and snow caves during an ascent of Denali.

Concentrations measured at altitudes between 2000 and 5200m usually exceeded the U.S. Environmental Protection Agency's 1-hour standard of 35ppm, reaching a mean value as high as 165ppm in a snow cave. Based on the data from this preliminary study, the use of a larger vent hole, approximately the size of a ski pole basket, appears to provide enough ventilation to maintain CO levels at more acceptable levels in snow caves and igloos.

INTRODUCTION

Ever since the early polar explorers began using portable kerosene stoves to cook and melt snow for water, numerous accounts of people being poisoned by the exhaust gases from such stoves have been published (1-3). One such account comes from the journey of Stefansson to the North Pole. He reported that while using a Primus stove (a small kerosene stove) in a snow house, the walls "became iced and impervious to gases," whereupon two of his companions collapsed in the house while he and the fourth member of the party managed to extinguish the stove and escape before collapsing: "An hour later three of the party members were well enough to go into the house again..." (1).

In the modern era, we have the advantage of considerable research on topics such as emission rates of carbon monoxide (CO) from stoves and lanterns (2,4), the concentrations of CO that can be found in tents and snow houses (1-5), and the amount of carboxyhemoglobin (COHb) found in the blood after carbon monoxide exposure (3). The acute health effects of exposure to carbon monoxide are well established (6). Most recently, Cohen (4), using a combination of laboratory work and computer modeling predicted that high levels of CO could be attained in tents when a cooking stove was used even for periods less than 30 minutes if ventilation were inadequate.

Despite considerable knowledge in this area, morbidity and mortality from CO poisoning persists on Mt. McKinley (Denali). In 1986, two Swiss climbers died on Denali while cooking in a tightly sealed tent at 4400m, ironically, only a few meters from the medical research camp. The purpose of this study was to better define the exposure to CO on Denali under typical mountaineering conditions in typical shelters and to test various methods of reducing the hazard by changing shelter ventilation.

Measurements were made during and after the use of the stove for meal preparation. The data indicate that CO exposure may reach toxic levels in all types of shelter and that adequate ventilation in snow shelters can be established by larger vent holes.

METHODS

Two of the authors (WT and SM) made the measurements during a climb of Denali in June of 1985. Measurements were made in three types of shelters used for mountaineering: tent, igloo, and snow cave (Figure 1). Because climbers often encounter temperatures well below freezing and strong winds, these shelters are designed to be small in volume and to have low ventilation rates. The tent was nylon, designed for two persons, with double-wall construction and a vestibule for cooking that was open to the inside of the tent. Igloos were made with cut snow blocks, and the cooking and sleeping level was higher than the entrance. Snow caves were also constructed with the living area higher than the entrance to help maintain warmth.

Carbon monoxide (CO) measurements were made using a calibrated CO monitor (Gas Tech Portable, Model CO-82). The instrument was zeroed before and after each series of measurements. A calibration standard of 180ppm CO was used at 90m, 2600m, 3400m, and 4300m above sea level to define a relationship be-

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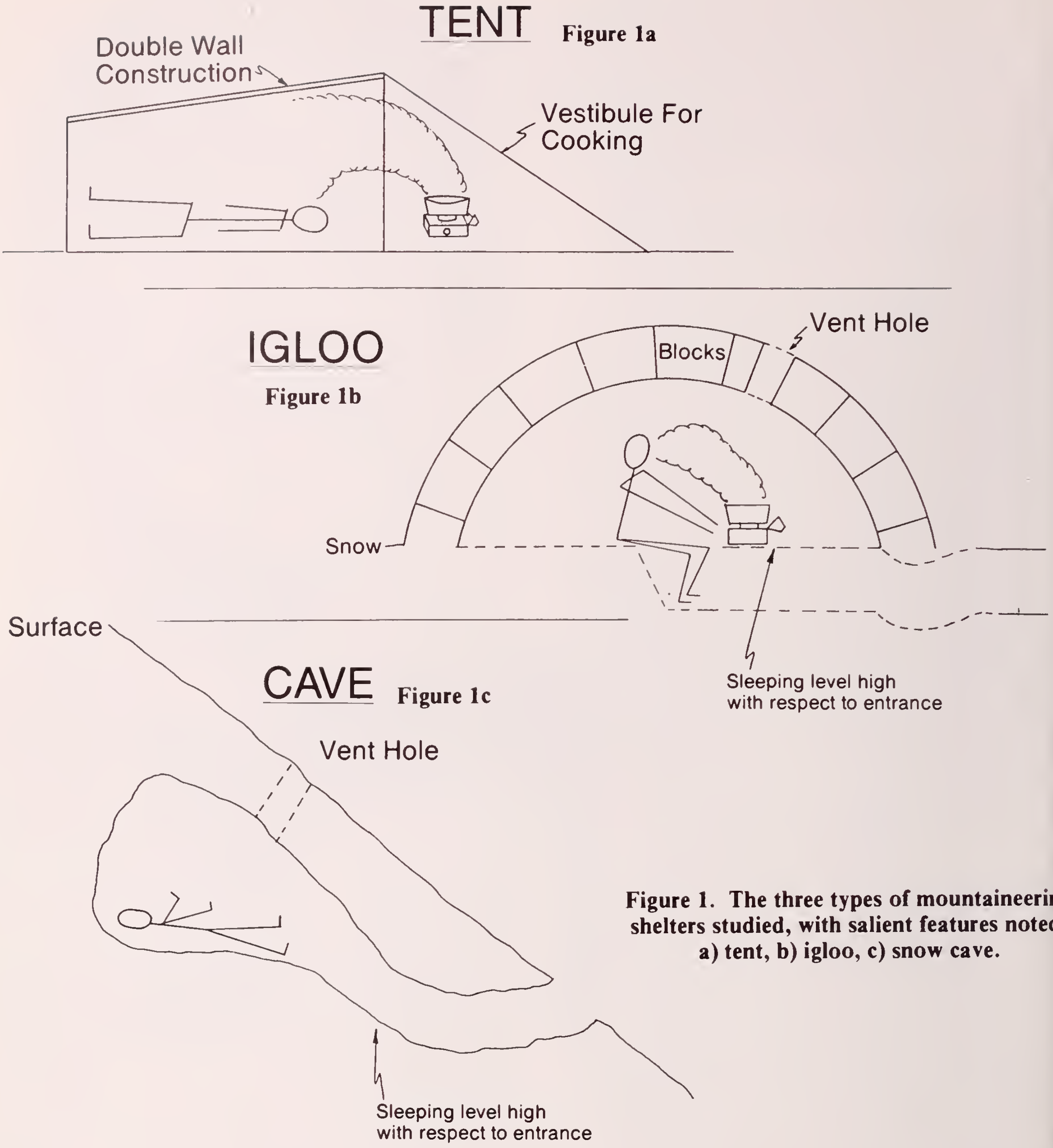

Figure 1. The three types of mountaineering shelters studied, with salient features noted: a) tent, b) igloo, c) snow cave.

tween the instrument's response to the standard and the altitude. When plotted, an altitude correction function was derived and applied to the data. (The instrument reads low at high altitude due to decreased partial pressure of gas.) This was preferred over adjusting the instrument at each altitude. Readings of the CO instrument were recorded at approximately 10-minute intervals during monitoring.

Three different stoves with similar fuel consumption rates were used during the testing. These were an Optimus 111B, an Optimus 8R, and a MSR Firefly. All stoves burned white gas and were in good operating condition. None had special jets for high altitude, and no attempts were made to alter the air/fuel ratios. The maximum fuel consumption rate for two of the three stoves (111B and 8R) has been measured to be approximately 210 grams/hour and is estimated to be less for the Firefly.

Measurements were made during the preparation of the meals in each of the dwellings. These periods typically lasted between one and two hours and consumed 150–300 grams of fuel. The detector was located in the breathing zone of the occupants, approximately 1.5 to 2 meters horizontally from the cookstove and approximately 1 meter vertically, depending on the particular shelter. Efforts were made to standardize this sampling location. CO measurements were also made after the stove was turned off to calculate ventilation rates. Assuming exponential decay, the ventilation rate (air exchange rate of the dwelling) is the slope of the logarithm of CO concentration plotted against time. A more in-depth study of this method and its assumptions can be found in American Society of Testing and Materials (ASTM) (7). In snow caves, all measurements were repeated with different sized ventilation holes. Ventilation area was changed in the tent by partially opening or closing the zippered door. The reported CO concentrations have been corrected for altitude.

RESULTS

Tent

Table 1 gives the data that was taken inside the tent at 2300m using the 111B stove. During the last two runs, the CO concentrations were notably higher than the first two runs and were above the toxic level (50ppm). The outdoor wind speed during the first run was 7m/s, while there was no detectable wind during the following measurements. This explains the high ventilation rate in run 1 as measured by CO decay rate.

Igloo

The results from using the 111B and 8R stoves in the igloos at 2600 and 4300m are presented in Table 2. CO reached toxic levels in all tests when the stoves were burning for more than fifteen minutes. The first test was made while the snow blocks were still fresh and porous, which may have led to the relatively high air ventilation rate (air exchange rate) observed. The last three runs were made in igloos that had relatively less permeable walls due to glazing and accumulated snowpack. There was a decrease in ventilation rate for the later tests.

Snow Cave

Table 3 summarizes the results for the snow caves at 5000 and 5300m using the 8R and MSR stoves. While in these snow caves, the highest CO concentration was recorded (190ppm). Therefore, we investigated the relationship between concentration and the vent size.

Vent Area Versus Ventilation Rate

In a snow cave, vent holes of different sizes were made to test the relationship between air exchange rate and the size of the hole. The size of the vent area does not include the opening at the cave entrance, since the floor of the cave is higher than the entrance. Three basic sizes were tested: an ice axe shaft hole (approximately 10cm^2), a ski basket hole (approximately 50cm^2), and a hole made with the handle of a shovel (approximately 80cm^2). The relationship between vent area and the ven-

Table 1

TENT				
Stove Type	111B	111B	111B	111B
Altitude (meters)	2300	2300	2300	2300
Delta temp. (degrees C)	11	6	8	6
Wind Speed (m/s)	7	none	none	none
Burn Time (hrs)	.75	.5	.5	.5
CO conc (ppm)				
# Points	10	5	6	6
Max.	40	40	80	110
Mean	25	35	50	70
Min.	15	25	15	55
Gross Ventilation (cm^2)	4500	19400	2600	19400
Ventilation Rate (air exchange/hr)	150	65	13	38
# of points in decay curve	3	15	10	9

tilation (air exchange) rate is shown in Figure 2. In this limited data set the ventilation rate closely correlates with the gross vent area ($R^2=0.903$). A hole of 50cm^2 more than doubled the ventilation rate, and also kept CO concentrations below the EPA standard of 35ppm for one hour.

Air Exchange Versus Dwelling Type

Air exchange rates in the three shelters were determined by decay of CO over time with the source off. Figure 3 shows typical decay rates for the tent, the igloos, and the snow caves with holes. For the tent, the entrance door was half open. Even without perceptible wind, the ventilation rate was calculated at 65 air exchanges/hr (ACH). The inside vs outside temperature difference was about 6°C. CO concentrations reached background ambient values in three minutes. For the igloo, with a vent hole of approximately 45cm^2, a typical air exchange rate was five air changes/hour, and the CO concentration had not reached ambient levels after 30 minutes. For the cave with a vent hole of approximately 20cm^2, a typical air exchange rate was 3.6 air changes/hour, and after 45 minutes the CO concentration was still relatively high.

DISCUSSION

The observations and experiments performed on Denali between 2000m and 5200m were made under typical snow camping conditions. The stoves and shelters are all common to mountaineering. The snow burn times were actual meal or water preparation times required to support two or three climbers. It is important to note that 8 of the 13 sets of measurements of CO exceeded 50ppm during stove use. Our data, though limited, indicate that mountain climbers on Denali who cook in shelters may be at risk of acute carbon monoxide poisoning and that increasing shelter ventilation effectively reduces the risk.

Average CO concentration ranged from 15ppm to 165 ppm, varying primarily with shelter ventilation rates but also with fuel consumption and stove type. The air

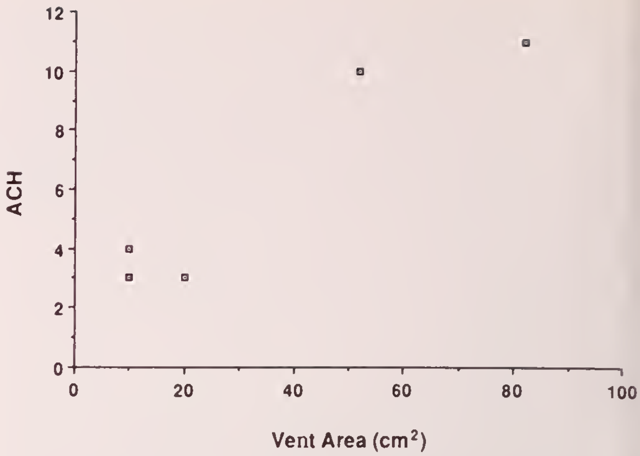

Figure 2. Air exchange rate (ACH) vs. size of vent area in a snow cave. A vent greater than 50 cm sq resulted in higher exchange rates.

Table 2

	IGLOO			
Stove Type	111B	111B	8R	8R
Altitude (meters)	2600	4300	4300	4300
Delta temp. (degrees C)	17	23	23	23
Wind Speed (m/s)				
Burn Time (hrs)	1	1	.5	.25
CO conc (ppm)				
# Points	8	14	5	4
Max.	85	140	80	40
Mean	80	120	70	35
Min.	70	80	60	30
Gross Ventilation (cm^2)	none porous	45 glazed walls	45 glazed walls	45 glazed walls
Ventilation Rate (air exchange/hr)	13	5	4	14
# of points in decay curve	17	22	10	10

exchange rates were determined to be lowest in igloos and snow caves where the walls are more impervious to air flow and ventilation holes limited. We confirmed similar CO levels and ventilation rates during March 1986 in snow caves at 1000m and 1500m in the Presidential Range of the White Mountains of New Hampshire (unpublished observation).

The plot of the CO concentration decay suggests that the mechanism for ventilation in the snow caves and igloos is relatively similar, while the tent may differ. Wind is one factor expected to exert much more influence on ventilation in a tent compared to snow shelters, especially once an interior ice-glazed surface is formed. It should be noted that during the first experimental run inside of the tent, a wind of approximately 7m/s was blowing outside. This run resulted in an air exchange value of 150 per hour. Subsequent measurements were in calm conditions, and air exchange rates were closer to the other two shelters but still generally higher for the tent. The much larger vent area of the tent appears to be a distinct advantage. It should be noted that tightly sealed tents, however, could also form an ice glazed interior surface in the absence of wind or be covered with snow on the outside; even tents have the potential for low air exchange rates, as demonstrated by the recent deaths on Denali.

The effectiveness of using larger vent holes in snow caves is an important finding. We found the relationship between the air exchange rate in the snow cave and the vent area was relatively linear, implying only a small contribution to the ventilation rate was made by diffusion of air through the semi-porous snow and ice. In order to make a rough estimate as to what vent size would be required to keep the CO concentration below a certain level, the CO concentration was also plotted against the vent size. We recommend a minimum of 50cm² vent in a snow cave, which seems to be adequate even if the snow walls are glazed.

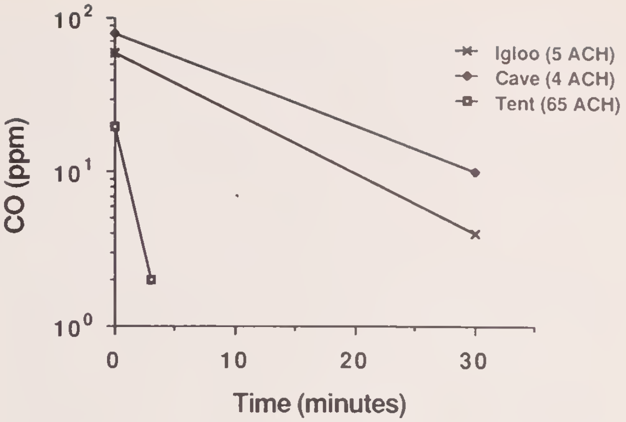

Figure 3. Carbon monoxide concentration in parts per million over time for the three different shelters. The shelter with high air exchange rate (ACH) had the most rapid decline in CO after the stove was turned off.

The emission rate (or source strength) of the cooking stove also influenced the level of CO. Based on fuel consumption rates determined for the 111B and 8R and boiling times for all three stoves, it appeared that the MSR Firefly had a lower fuel consumption rate and

Table 3

SNOW CAVE					
Stove Type	8R	8R	MSR	MSR	8R
Altitude (meters)	5000	5000	5000	5300	5300
Delta temp. (degrees C)	8	11	14	11	5
Wind Speed (m/s)					
Burn Time (hrs)	1 hr.	1	2	1	.25
CO conc (ppm)					
# Points	10	6	11	4	3
Max.	190	35	95	65	30
Mean	165	30	70	50	28
Min.	70	25	60	38	25
Gross Ventilation (cm²)	11	52	20	11	81
Ventilation Rate (air exchange/hr)	4	10	3	3	11
# of points in decay curve	18	4	6	11	5

thus possibly a lower emission rate.

The concentrations of CO recorded in the various shelters are high enough to produce significant toxicity. Of the 13 total experiments, eight showed mean concentrations of 50ppm or higher. Fifty ppm CO results in a carboxyhemoglobin level of about 8%, assuming the kinetics of uptake and equilibration remained the same as sea level. There is some evidence, however, that CO uptake is increased because of the hyperventilation induced by the hypoxia of high altitude (8,9). Although 8% COHb is not considered toxic at sea level, such a level will exacerbate the altitude hypoxia. The normal arterial oxygen saturation (SaO2%) of a climber on Denali after two days acclimatization to 4400m is 80%. A decrease in oxygen carrying capacity of 8% to an effective SaO2% of 72% due to COHb would result in a very significant drop in oxygen transport, rendering the climber at a "physiologically" higher altitude and could easily precipitate acute mountain sickness. In 1985, two Denali climbers required rescue because of severe acute mountain sickness induced by CO exposure (10). Low-level CO exposure may thus contribute to altitude illness. Further evidence for an additive effect of CO and altitude hypoxia was a study by Vollmer *et al.* (11). Four of seventeen healthy subjects at a simulated altitude of 4725m collapsed when COHb reached 9% to 19% (9), values not associated with collapse at sea-level. The EPA has more recently reviewed the combined effects of CO and altitude (12).

The higher CO concentrations recorded in our study are clearly in the range to cause CO toxicity, depending mostly on the exposure time. Symptoms of CO poisoning are identical to those of acute mountain sickness; i.e., headache, nausea, dizziness and lassitude. Therefore, differentiating the two may be impossible. Also, because of the lower inspired oxygen pressure, the rate of elimination of CO from hemoglobin may be impaired.

Two climbers found dead of CO poisoning from cooking in a tightly sealed tent on Denali in 1986 had COHb levels of 65.6% and 56.9% at autopsy, levels considered lethal even at sea level. Although the interactions of CO and altitude hypoxia need further investigation, it is clear that CO poisoning poses a serious risk when using stoves in shelters at high altitudes.

SPECIFIC RECOMMENDATIONS

As a result of the measurements obtained, the authors recommend that tents, igloos and snow caves always be vented when combustion appliances are used. We suggest that mountaineers adopt the practice of creating a vent hole in igloos and snow caves that is at least the diameter of a ski pole basket whenever a stove is in use, especially at high altitude. This vent should be located in the vicinity of stove operation and as high as possible in the dwelling. To maintain vital warmth offered by the shelter without risk of physical impairment from CO poisoning, the vent hole may be plugged with snow after finishing use of the stove. Using this method, the authors on this McKinley climb were able to maintain temperature within properly constructed igloos and caves in the range of 15 to 20°C above ambient except when the vent was open.

In light of the recent mountaineering deaths and illness attributable to CO poisoning and increasing activity of high altitude mountaineering, further research is needed in this area. Altitudes above 4000m are sufficiently hostile without the added danger of carbon monoxide.

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Early Alaska Mental Health

by Thayne I. Andersen, M.S.W.[1]

INTRODUCTION

It is easy to think that depression, paranoia, suicide, schizophrenia and other mental illness conditions (because of recent classifications by medical researchers and clinicians) are only modern–day situations. Certainly, securing adequate treatment for modern mental illnesses often seems to be an impossible task for today's medical personnel, but their efforts are not distinctively new.

Serious mental health problems are not just a 20th century phenomenon in Alaska. Various types of mental health issues were handled by early United States medical and governmental authorities in territorial Alaska. Even prior to the United States possession of Alaska, there were mental health problems here. The Russian–American Fur Company employees also had mental health problems as did the families of Alaskan Natives prior to the 1867 transfer of Alaska to the United States from Russia.

Alaska Natives

Prior to the arrival of other ethnic groups, Alaska's Native inhabitants had to deal with behavior deviating from the accepted "normal" behavior within their own family, clan or community. Without a written Native language, there is little that a modern Alaska researcher can conclude about how deviant behavior was handled by Natives. His only option is to explore early Russian writings and draw conclusions from them.

Given the wide variety of Native cultures in Alaska, there was probably a considerable degree of difference among the various Native groups in how they dealt with mental health problems and social deviance in pre–anglo Alaska. In many Alaskan Native groups, the treatment and explanation of mental illness was rooted in magic and the power of the shaman (1).

One Native group, the Tlingits (the Russians referred to them as "Kolosh") were reported sometimes to believe that when an adult married person committed suicide, his/her spouse "drove" him/her to it. The remaining spouse might be required by the deceased person's relatives to pay them for this wrongful act. This would have been particularly true if the suicide occurred after the deceased drank alcohol supplied by the surviving spouse.

Murder committed against a spouse or anyone else in the community had to be atoned for. Insanity, temporary or otherwise, was not considered a justification. The severity of the punishment demanded by relatives of the deceased was based on the social rank of the deceased and his/her importance, not the mental state of the assailant. This atonement was not just required of the person accused; his family and relatives might also be held responsible. In the case of a husband killing his wife (or being blamed for her suicide), it was possible that his sister—not he—might forfeit her life to pay for his uncontrollable temper or rage.

The Russians and the early Americans noted that the Natives often tried to find causal relationships when mental impairments, disease, hysterical reactions, or illness were manifest. Like the whites of early New England, the Natives thought they saw a relationship between these events and a type of witchcraft in the form of spells cast by someone who had a grudge against the ill person. The obvious treatment, then, was not to try to work with the ill person, but to try to find the person responsible for casting the spell and try to get him/her to free the patient from the spell that was afflicting him.

The Russians

When the Russians came to the shores of Alaska, they brought with them their own ideas of justice as well as their concepts of what constituted mental illness. Our current system of classifying mental illness was not available to the Russians or to the early U.S. officials for that matter. But there are instances of "insanity" and alcohol–related problems including depression among the Russians. One author, in writing about Alaska, stated his opinion that "ultimate frontiers draw the psychopaths" of society (2). If this were true, then the percentage of Russian fur trader/frontiersmen (called "promyshlenniki") who had mental problems was high.

These rough fur traders were often the outcasts of Russia: those who had been exiled from the eastern Russian cities or were running away from the law. They were taken—often against their will—to a far–off land to work as virtual slaves to a monopolistic company. In a letter to the Directors of the Russian–American Company, Rezanov wrote:

> "Most of the men who come here are depraved, drunk, violent and corrupted to such an extent that any society should consider it a great relief to get rid of them (3)."

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About the only medication available for mental or physical illnesses to the early adventurers in Alaska, whether they were Russians, local Natives or Americans, was a strong self prescription of a cure-all containing enough ethyl alcohol to help them temporarily escape reality. Sometimes this was easy enough to do by brewing a beer called "quass," or, if lucky enough, they could purchase or make distilled liquor. One could get drunk with either product, though it was quicker and easier with vodka or whiskey.

A good example was Alaska's Russian "Governor," Alexander Baranof. Even though he was a capable manager of early Alaska, he showed signs of alcoholism during some of the time he lived in Alaska and was claimed to have been in a deep depression for one full winter after an attempt on his life in Sitka. During the winter of 1809–1810, he could be roused only to look after the major chores of his office. His medicine of choice was rum which he distilled in a private still to provide a reliable supply of medication for his "illness" (4).

On November 28, 1868, shortly after the assumption of U.S. jurisdiction over Alaska, the military commander of Alaska, General Jefferson C. Davis, received a request from the former Russian Governor to allow an "insane" Russian-American (actually, the diagnosis was "non compos mentis") to be sent back to Russia with Prince Matsoutoff. Even though the patient was probably technically an American citizen, General Davis quickly complied with the request since he had no authority to do anything else with him (5). Supporting this request was the medical opinion of the U.S. Army physician at Sitka, Dr. Alexander H. Hoff. Before making his decision, Davis also interviewed the patient and came to the same diagnosis.

U.S. Jurisdiction of Alaska

At the time the United States took possession of Alaska in 1867, only one mental hospital had been established in the entire United States. It had been made possible by an act of Congress on March 3, 1855 and was located in Washington, D.C.. It was called the "Government Hospital For The Insane." An act of June 23, 1874 authorized the U.S. Attorney General to contract with state asylums for the care of insane persons. But since Alaska was not yet a state and in fact had no kind of government, those Alaskans needing care were not eligible for admission. On August 7, 1882, an act authorized the Secretary of Interior to commit Alaskans to the Government Hospital For The Insane (6). No one from Alaska was ever committed to this hospital 3,500 miles away from Alaska.

As far as I can determine, the first person to be transported from Alaska to the states prior to 1901 was a man by the name of Frank W. Aaron. In 1884, Mr. Aaron was being held in the Sitka jail when U.S. Marshall Hillyer was instructed by the court to transport the "insane" patient to the California State Hospital For The Insane at Napa. This hospital was designated due to the great distance of the Washington, D.C. Hospital from Alaska. Apparently, some kind of contract or agreement was worked out with this hospital by the Attorney General in anticipation of future referrals, but as it worked out, Mr. Aaron was the only patient officially transferred out of Alaska till 1901—about 17 years later (7).

There were three problems, in addition to the distance, that stood in the way of using the Government Hospital For The Insane:

1. To qualify for admission, the patient could not just be diagnosed as "insane" by a physician (even if one were available), but he had also to be a criminal who had been tried and found guilty of violating a federal law and then sentenced to the hospital under U.S. statutes. Until 1899, since most offenses in Alaska were charged under Territorial codes applying to Oregon, the majority of prisoners were thus ineligible.
2. There were no provisions for care of mentally ill persons who were not charged with violating the law.
3. Even in those infrequent instances when admittance might have been authorized, the Attorney General was not empowered to expend funds for the transportation of the prisoner to the hospital.

The Criminally Mentally Ill

If the biggest drawback to getting hospitalization was the lack of a criminal conviction, then Frank Fuller should have been the next case to be hospitalized. In 1886, Fuller shot and killed a Catholic bishop by the name of Charles Seghers who was doing missionary work in Alaska. There was ample evidence that Fuller was mentally ill at the time of the murder.

About five weeks before his death, Bishop Seghers wrote in his diary: "...peculiar conversation with brother in which for the third time he gives evidence of his insanity (8)."

Fuller was overly suspicious of people, thinking that everyone was out to hurt him. Six months after the murder, while enroute to Sitka for trial, Fuller admitted to the murder saying, "God told me to do it." In today's jargon he would likely be labeled as a paranoid schizophrenic. Fuller was tried for murder. His defense attorney, against the prisoner's wishes, entered a plea of insanity (9). He was found guilty of the reduced offense of manslaughter and was sentenced to imprisonment at McNeil Island. Without adequate facilities to treat mental illness, the penitentiary released him unconditionally nine years later.

Role of U.S. Marshall

In early Alaska, the only person available representing government authority often found that he had been given more work than he had bargained for. The person who was required to find resources for mentally ill Alaskans prior to the gold rush, since the earliest physicians came and left with the military, was the U.S. Marshall.

A good example of this was the case of George H. Berry in 1890. The U.S. Marshall at that time, Orville T. Porter, determined that Mr. Berry needed to be hospitalized for a type of progressive mental illness possibly caused by syphilis. Marshall Porter requested of the U.S. Attorney General's Office that Mr. Berry be admitted to the Hospital in Washington, D.C.. Since Berry had committed no crime, Marshall Porter was advised that there were no provisions under the law that would authorize such a transfer. In typically slow-moving bureaucratic style, the Marshall was advised by the U.S. Attorney General that the matter would be referred to the U.S. Senate Committee on the Territories for possible legislation that could resolve this issue for Mr. Berry. The Attorney General then advised the Marshall: "In the meantime you will care for said Berry in the best manner possible (10)."

Mr. Berry was removed from the jail and placed in separate quarters under the care of a male nurse—at the personal expense of the Marshall. Mr. Berry died there several months later, still waiting for approval by an act of the U.S. Congress to be admitted to a hospital.

It was often the case that the law enforcement officer in early Alaska was forced by his own generosity to support mentally ill citizens who had not committed a crime, since these types of expenses were not recognized by the Attorney General as authorized expenditures.

Another example of how those sworn to uphold the law had to battle the bureaucracy was an event that happened the year after Mr. Berry died. In 1891, Marshall Porter detained in his jail another patient whose only offense was that he was "insane." This patient was a well-known drunk named Michael Travers. Marshall Porter quickly wrote letters to his superiors asking for help in getting Mr. Travers to a mental hospital.

The reply this time came from the U.S. Department of Justice. It reaffirmed the fact that the law had made provisions for the mentally ill in Alaska to be hospitalized only if they had violated federal law. Marshall Porter was told to figure out a way to bring criminal charges against his prisoner, a successful conviction on which would qualify Travers for admission. In summary, the agency responsible for upholding the law suggested that manufactured charges be brought against a person who had broken no law, while he was in jail due to being mentally incompetent, so that he could qualify for admission to a mental hospital (11)!

Until physicians came to live in Alaska after the gold rush, the task of caring for the mentally ill (at least the mentally incompetent) fell, by default, to the U.S. Marshall. He acted as their protector, social worker and placement officer. It is a tribute to these early police officers in Alaska that they did as well as they did with their many roles.

Bureaucracy Isn't only a Modern Phenomenon

When today's mental health workers feel down and out about the vast amount of red tape they have to go through, they would do well to know what the early U.S. Marshall had to face. To obtain the necessary funds to escort a patient (or prisoner, for that matter) a marshall was required, after receiving official approval for the transfer, to have his vouchers certified in open court. They then had to be approved personally by both the Territorial Governor of Alaska and the U.S. Secretary of the Interior. The vouchers were then transmitted to the Secretary of the Treasury. If the documents were in order, funds would be disbursed to the Attorney General, who would in turn advance money to the marshall to pay for the transportation to Oregon and return. In addition, the Governor required that a permit for removal be obtained from his office in advance of the removal. After 1901, "insane" patients were transferred quite regularly from Alaska to Oregon (mostly to Morningside Hospital) notwithstanding the interminable delay between adjudication and institutional commitment (12).

After the Territory of Alaska built the first Pioneer's Home in Sitka in 1912, several admissions were made to this facility of patients who, though not "insane," were suffering from various kinds of mental illnesses. The records of the Sitka Pioneer Home show several instances of suicide among their early patients (13).

After World War II, the military hospital on Japonski Island (across a narrow channel from Sitka) was designated as a facility to be used by Alaskan Natives for the primary treatment of tuberculosis. It is often referred to by early Sitka newspapers as "The Sanitorium," which has connotations of being used for the treatment of chronic illnesses of various kinds, including mental health patients (14).

SUMMARY

It is easy to think of mental health patients and problems as modern-day happenings to the exclusion of our past. Certainly, no one disputes the fact that it is often difficult today to get many patients the kind of treatment their diagnosis demands. Alaska's historical heritage shows us a perspective, however, that is important to medical professionals who are working with the mentally ill. This article has shown a little of what happened in early Alaska as efforts were made to get treatment for these patients. In a growing territory, it was often excruciatingly difficult to work with systems and laws that were never intended to deal with far off

Alaska.

Further research could well be done on the types of mental illness referrals that went to Morningside Hospital in Oregon after 1902, and what the physician's role was in these referrals.

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12. Murton, T. 1965;90-91 Several other cases are documented in this study of mentally insane patients being kept in jails or boarded at the personal expense of law men.
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14. I could not verify that there were any referrals to this hospital specifically for mental illness, however.

A review of Alaska historical records was made as part of research in preparation for a soon-to-be-printed book about Alaska's early alcohol history. A large part of this article is based on a thesis, "The Administration of Criminal Justice in Alaska, 1867 to 1902," by Thomas Murton, (1965), University of California, Berkeley.

Toxic Wastes and Health; How Real is the Problem?

by Samuel Milham, Jr., M.D.[1]

Hardly a day passes without a newspaper or TV newscast proclaiming a dire warning of the health hazards posed by toxic wastes. In the past year, locally, EPA has claimed that there are excess lung cancer deaths in Tacoma residents due to arsenic emissions from the ASARCO copper smelter (1), and EPA workers were shown in the Seattle newspapers taking environmental samples from Gasworks Park in North Seattle while attired in protective "space suites" (2). A federally funded clean-up is under way at the Western Processing "hazardous waste" site in Kent, Washington, and parts of Puget Sound have been closed to sports and commercial fisheries due to "carcinogenic" residues being discovered in bottom fish and sediments (3). Food has been removed from local supermarket shelves due to low level contamination with ethylene dibromide, groundwater in various parts of the state is contaminated by industrial wastes, and most urban and surface water samples contain known animal carcinogens.

Nationally, Times Beach, Missouri (4) and Love Canal (Niagara Falls), New York (5) have been viewed by government as being a serious enough threat to human health to require condemnation proceedings and relocation of exposed families.

If there is potential public health impact when these episodes hit the local news, my telephone usually rings. In the 19 years that I've been answering the phone, there have been very few instances where I felt that there was compelling evidence that the health of people was being compromised by community exposure to toxic wastes.

At the outset let me state that humans have been killed and made seriously ill by community exposures to toxic wastes. Industrial waste water containing methyl mercury was discharged into Minamata Bay in Japan. It contaminated the aquatic food chain and caused severe illness in people who ate contaminated fish (6). Some infants exposed *in utero* were damaged for life. There are other examples of mortality and morbidity associated with accidental contamination and intentional adulteration of food stuffs.

I also believe strongly that clean air and water are better for human health than contaminated air and water. This conviction notwithstanding, I find little evidence to support the view that the public's health is compromised by most of the toxics-related episodes which receive media attention.

For a toxic substance to have a health impact, it must be delivered in a *sufficient dose* by an appropriate route. People are exposed by eating, drinking, or inhaling the toxic substance. Uncommonly, skin exposure can be a factor, and in the case of radioactive wastes, just being near an emitter of ionizing radiation can be hazardous. From my experience, the dose of a toxic substance delivered in most community exposure situations is insufficient to cause disease. We can measure contaminants in water and air in minute amounts. Parts per billion (PPB), trillion (PPT), and quadrillion (PPQ) are very small amounts, and our analytic techniques seem to be getting better with time.

Most of the toxic materials of concern in community exposures are present in air and drinking water in such small amounts that dose levels of acute toxicity are rarely achieved. The critical question is whether chronic, low dose exposure has an effect on health. I submit the best way to study the problem is to examine populations which have chronic intermediate and high dose exposures to the chemicals of interest. This, of course, is in the occupational setting.

The occupational setting can tell us a lot about how humans respond to various chemical (and physical) exposures. A work force is identifiable, can be followed over time—even to death, and exposures can be measured or estimated. Extrapolation to the community from the workplace has some problems: workers are most often healthy adult males, and they are exposed for 8 hours, 5 days-a-week. In the community, exposures are for 24 hours, 7 days-a-week, and infants, pregnant women and chronically ill people may be exposed.

The other way to assess potential human risk to toxic chemicals is to conduct formal animal toxicological assessment. In this case, rat and mouse results have to be extrapolated to humans. A critical and insoluble problem with both human and animal dose-response data is extrapolating from the high and intermediate dose levels in experimental and occupational studies to the very low dose levels seen in community exposures. The extrapolation procedure boils down to taking an educated guess. It is almost never possible to do animal studies or to find occupational groups which can give us data points on the low end of the dose-response curve where most community toxic exposures are. My operational rule of thumb is that if an occupational cohort with very heavy chemical exposures does not show a health problem, then community exposure levels will probably not show any health effects.

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[1] Chronic Disease Epidemiology, Department of Social and Health Services, LP-15, Olympia, Washington 98504. This paper originally was presented on June 21, 1984 to the Washington State Trial Lawyers Association.

Table 1. Mortality in Workers
ASARCO Copper Smelter, Tacoma, Washington

Men who worked a year or more, 1940–1964, followed to 1976 (from Enterline & Marsh).

Cause of Death	Deaths Observed	 Expected[1]	 SMR[2]
All Causes	1,061	1,028.5	103.2
All Cancers	231	186.9	123.6*
Respiratory Cancer	104	54.9	189.4*
Stroke	91	81.7	111.4
Heart Disease	412	445.6	92.5
Respiratory Disease	60	55.3	108.6
External Causes	81	86.0	94.2

1. Based on the U.S. Sex-, Age-, and Calendar-specific death rates

2. $SMR = \frac{\text{Observed Deaths}}{\text{Expected Deaths}} \times 100$ **= Standardized Mortality Ratio**

***** $P < .05$.

For example, although EPA bought out Times Beach, Missouri because of contamination of soil with dioxin, I do not think there was much of a health problem. There has been no chloracne, a skin condition which follows moderate exposure, and no one has been ill with the possible exception of one case of hemorrhagic cystitis in a young girl (4). The occupational literature is quite reassuring. Men who manufacture 2, 4, 5-T (7,8) and trichlorophenol and men exposed to dioxin in a trichlorophenol process accident (9) show no excess total mortality or cancer mortality in spite of the fact that many of them had chloracne and other medical problems consistent with high dose dioxin exposures. Additionally, in 1976, at Seveso, a city near Milan, Italy, a factory accident released a large cloud of TCDD (tetrachlorobenzo P-dioxin) into the community. Animals died and chloracne cases developed. But in the 10 years which have now passed, very little in the way of detrimental health effects have surfaced (10).

Although media coverage suggested dire health problems at Love Canal, to my knowledge no adverse health effects due to toxic exposures have resulted. I had the good fortune to sit on an advisory committee which examined the accumulated Love Canal data. There was little evidence of human exposure to toxic chemicals and no convincing evidence of increased morbidity or mortality. This is not surprising since measured pollutant levels in the air were very low (11). A recent report (12) shows that Love Canal residents do not have chromosomal aberrations as had been widely publicized earlier (13).

In the early 1970s, I began to look at people who resided around the ASARCO Tacoma Copper Smelter for possible adverse health effects of heavy metals emitted by the smelter. The smelter is located in the town of Ruston adjacent to the City of Tacoma, Washington in an urbanized area. There had been early reports of chronic lead toxicity in children living around other lead emitting smelters (14). I was relieved that blood lead levels in Ruston school children were normal. However, urinary arsenic levels were high (15). This was not unexpected, since the Ruston Smelter processes copper ores which are rich in arsenic. In fact, the facility was the sole commercial source of arsenic trioxide in the United States. I spent the next couple of years looking for possible detrimental health effects due to this exposure. The following parameters were looked at and were found to be normal in Ruston school children.

1) Growth and Development
2) Blood Counts and Blood Chemistry (16)
3) Hearing (16)
4) School Absenteeism (16)
5) Academic and Physical Performance
6) Blood Pressure
7) Sister Chromatid Exchange.

Most importantly, no lung cancer mortality increase was seen in people residing in census tracts close to the smelter (17).

Arsenic is a recognized human carcinogen. It causes lung cancer in copper smelter workers with high arsenic exposures. Table 1 shows results of a mortality study done at the Tacoma Smelter (18). Lung cancer deaths are twice expected based on United States male rates, but total deaths are only slightly increased.

Table 2. Airborne Inorganic Arsenic (As) Levels Measured as Breathing Zone Time-Weighted Averages (19)

Department	N	As Concentration - $\mu g/M^3$ Range	 $\bar{x}$	 s
Yard, Mobile Equipment and Track Personnel	11	0.3-80	12.4	23.0
Crushing, Sampling and Martin Mill	28	1.5-110	13.1	20.4
Herreshoff Roasters	42	nd 820	169.5	195.4
Reverberatory Furnace	20	nd-190	43.6	42.6
Converters	20	15-820	160.4	247.3
Anode Furnaces	9	10-60	28.9	20.9
Refinery	15	4-19	9.27	4.67
Refinery By-Products	5	5-210	40.0	90.1
Nickel Plant	5	4-11	7.4	3.0
Arsenic Plant	16	100-1940	548.1	488.5
SO_2/Acid Plant	4	5-280	76.3	135.9
Main Cottrell	10	nd-6933	1162.7	2170.8
Boiler Room/Power Plant	15	1-160	61.6	51.6
Maintenance	72	0.7-5310	108.3	624.2

N = number of samples; $\bar{x}$ **= standard deviation;** $\mu g/M^3$ **= micrograms per cubic meter of air; nd = non-detectable (**$<0.1 \mu g/M^3$**).**

Table 2 shows results of a recent sampling of airborne arsenic by department within the Tacoma Smelter (19). Note that the average arsenic levels exceed the current OSHA standard (10 micrograms per cubic meter) everywhere but in the refinery and nickel plant. At the arsenic plant, the air levels were 50 times the OSHA standard and at the Cottrells, 100 times the OSHA standard. In eight departments, maximum concentrations over 100 micrograms per cubic meter were seen. By contrast, in the community, average arsenic levels in air are under one microgram per cubic meter with an occasional excursion to 10 micrograms (19).

Table 3. Respiratory Cancer Deaths
By Cumulative Arsenic Exposure
ASARCO Copper Smelter Workers
Tacoma, Washington

Cumulative Exposure Arsenic (micrograms per liter) (Urine-years) mean of interval	Deaths		
	Observed	Expected	SMR[1]
302	8	4.0	202.0
866	18	11.4	158.4
2,173	21	10.3	203.2
4,543	26	14.1	184.1
13,457	31	12.7	243.4

1. $\text{SMR} = \frac{\text{Observed Deaths}}{\text{Expected Deaths}} \times 100$ = Standardized Mortality Ratio

Table 3 and Figure 1 show dose-response data for worker arsenic exposure versus lung cancer mortality risk. Note that the relative risk of lung cancer is not much different at high versus low cumulative exposure levels (1).

EPA takes this type of occupational dose-response data and performs a "quantitative risk assessment"; i.e., they guess at what is going on at the low end of the dose-response curve and apply this to the community around the smelter to estimate that there will be X number of extra lung cancers in the community due to ASARCO's arsenic emission.

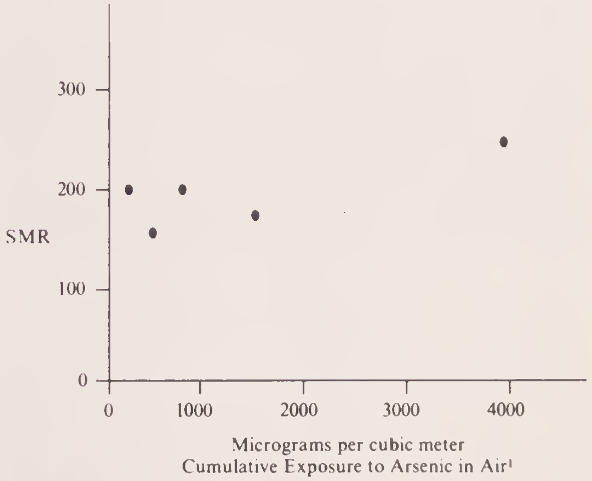
I have a number of problems with this process.

First of all, the EPA impact zone extends to more than 11 miles from the smelter. I cannot find any increase of arsenic in hair or urine of people residing over five miles from the smelter (15). It is hard for me to believe that doses of arsenic too small to detect on tissue (hair) or urine can be carcinogenic. More importantly, if extra lung cancer cases are being generated, why can't we detect them. Our calculations are that if one-half an extra case of lung cancer were being generated per year, we could detect the excess through our state death record system. So far, we have not seen an excess.

Although some would have you believe that toxic waste exposure is having a detrimental effect on the health of our nation as a whole, most of the standard indices of public health are improving; i.e., stillbirth rates, infant death rates, major congenital defect rates, and cardiovascular disease mortality rates are decreasing; life expectancy at birth is increasing. Although most national health measures are improving, small pockets of toxic exposure and resultant morbidity and/or mortality **do** exist and need to be recognized and remedied. The elimination of lead from gasoline is the sort of national action which will ultimately lead to improved health. In our society, life-style variables account for most of the observed morbidity and mortality. Even in the occupational setting, only a small fraction of the observed health problems can be traced to occupational exposure.

Let's clean up our dumps because they stink and are eyesores. Let's clean up our rivers because trout streams are nicer than open sewers. I'm afraid that if we were to clean up only those toxic waste sites which posed demonstrable hazards to public health, most sites would never be touched.

Figure 1. Standardized Mortality Ratios (SMRs)
By Estimated Cumulative Exposure
to Arsenic in Air
ASARCO Copper Smelter Workers
Tacoma, Washington



¹Estimated from mean urinary exposure index and .304, the estimated relationship between urine and airborne arsenic.

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Experience with Arthroscopic Anterior Acromioplasty

by Robert E. Gieringer, M.D.[1]

ABSTRACT

A series of 23 shoulders in 22 patients operated for Impingement Syndrome by arthroscopic acromioplasty after failed conservative treatment is presented with 18 good or excellent results (78%). Four fair results and one poor result were in workers' compensation patients. The complication rate was very low. This procedure is offered as an alternative to open acromioplasty. The concept of the Impingement Syndrome as a cause of shoulder disability is described. It is important that accurate pre-operative diagnosis be made to achieve satisfactory surgical results. Since there is less surgical morbidity, patients can be brought to arthroscopic surgery after unsuccessful conservative treatment at an earlier time than originally described by Neer, resulting in less interim disability.

INTRODUCTION

The concept of subacromial shoulder pain (Figure 1) has been described by many authors beginning with Codman in 1934 in his book, *The Shoulder,* which described disorders of the supraspinatus tendon. Bosworth, in 1941, described the "supraspinatus syndrome." De Palma has described the "painful arc syndrome" in his textbook, *Surgery of the Shoulder* (1). Finally, Neer described the "Impingement Syndrome" in his article in 1972 (2).

The impingement symptom complex primarily involves the coracoacromial arch (Figure 2) which intrudes upon the rotator cuff, subacromial bursa, and the biceps tendon (Figure 3). The acromioclavicular joint may be involved as a secondary factor in the syndrome. Complaints of subdeltoid bursitis and biceps tendonitis or pain with upward movement of the arm should raise a suspicion of the impingement condition.

Pain initially occurs with certain activities (Figure 1) as a result of mechanical contact of the structures within the arch against the arch itself. After a period of repetitive contact, pain may be lingering, and secondary changes (scar tissue, calcium deposits, bone spurs, rotator cuff tear) may occur. The impingement condition is primarily seen in those persons who have developed age-related changes in the shoulder. The stage is set by changes related to the vascularity in the supraspinatus tendon, scarring or thickening of the subacromial bursa, weakening of the rotator cuff lever arm, and hypermobility of the acromioclavicular joint. These might be a result of accumulated minor injuries, the summary of many years of active use, or both in combination.

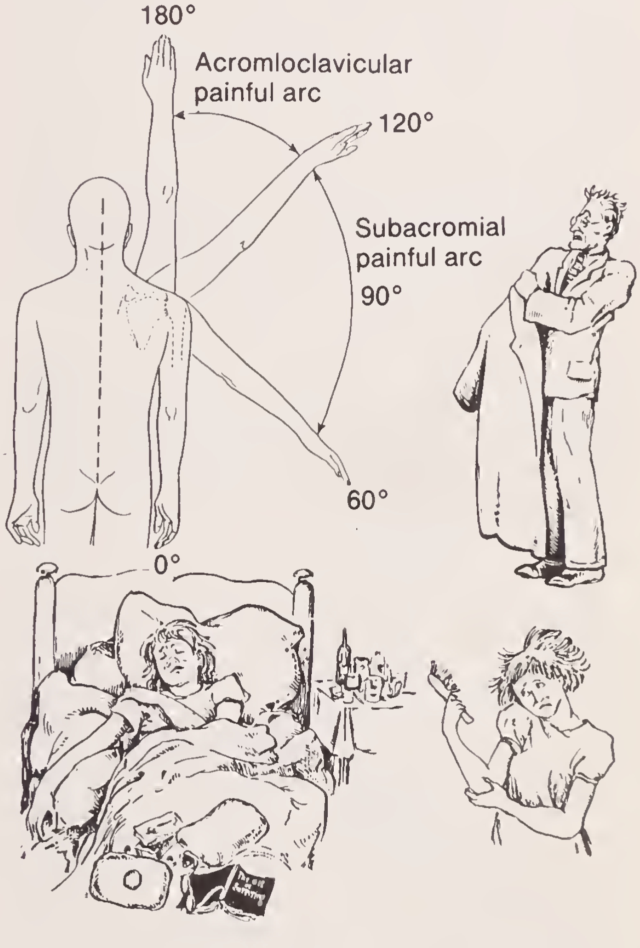

Figure 1. Modified from DePalma, 1983, and Moseley, 1953

The middle-aged patient is typically a person who continues to place the same demands on a body part which now has diminished capacity to meet those demands. An example of this might be a person with thickening of the subacromial bursa and supraspinatus atrophy who decides to wallpaper a bedroom over a weekend and then develops shoulder pain with racquet sports later that week. Bigliani has described three profiles (Figure 4) of the acromion as viewed through the supraspinatus outlet (3) in the general population which

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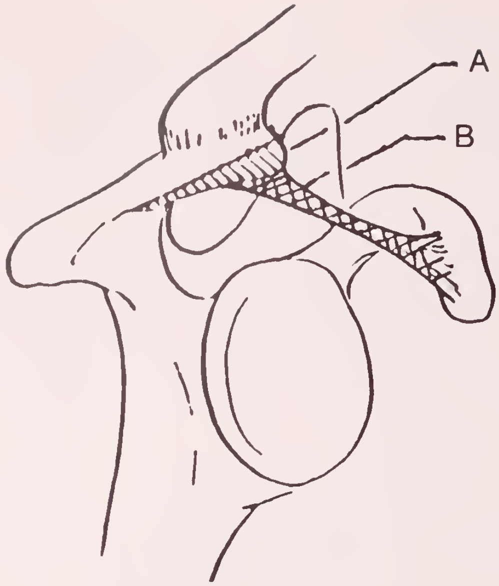

Figure 2. Structures of the coracoacromial arch A-Acromion, B-coracoacromial ligament. The shaded area of the acromion and the ligament (crosshatched) are resected when doing an acromioplasty.

may also affect the impingement condition (4). In this study the curved profile, type II, and to a greater extent the hook profile, type III, were associated with impingement lesions. Impingement in the young patient is commonly an overuse condition and will usually respond to rest or activity modifications.

Three theories of the etiologic factors have been promoted. The Mechanical Anatomical Theory, promoted by De Palma and Neer (1,5), describes an increase in the volume of the structures which occupy the subacromial space or a process that diminishes subacromial capacity, such as osteophyte formation. Another theory is the Vascular Compromise Theory of Kennedy and McNab, which describes an avascular area of the supraspinatus tendon which because of chronic mechanical impingement, leads to pain, irritation and focal cell inflammation (6). A third theory is a variant of the mechanical theory called the Biomechanical Basis described by Jacquelyn Perry (6). This describes kinesiologic factors limiting scapular rotation and uncoordinated muscular contractions in the shoulder girdle. According to this theory the scapula does not rotate up enough as the arm is abducted, leaving the acromion in the way as the humerus is raised. Or the supraspinatus and other rotator cuff muscles may develop relative weakness compared to the deltoid muscle. The pull of the deltoid vector then causes impingement against the acromion without the action of the rotator cuff to depress the humeral head, which would afford more clearance under the acromion. It is this latter theory upon which the use of physical therapy is based in the early treatment of the syndrome.

Encouraged by Codman's earlier statements, surgical treatment of the condition has been attempted by several authors over the years. This included acromionectomy, as described by Armstrong and Hammond, and partial lateral acromionectomy later described by McLaughlin and Smith-Peterson. The disappointing results of these procedures because of weakening of the deltoid muscle lever arm and displacements of the deltoid origin, bursal sinus tract formation, and persistence of residual symptoms led to the investigations by Neer which culminated in his classic report in 1972, describing anterior acromioplasty for the condition. In this surgical procedure only the anterior 1/3 of the under surface of the acromion and the coracoacromial ligament are removed using an osteotome or bone saw (Figure 5).

As initially described by Dr. Neer (Figure 6), there are three stages of the Impingement Syndrome: Stage I involves edema and hemorrhage of the supraspinatus tendon. Stage II is progression of the condition to fibrosis and tendonitis of the structures in the supraspinatus area. The final progression to Stage III shows bone spurs of the subacromial area and actual supraspinatus tendon rupture (rotator cuff tear). Dr. Neer's report in 1983 (5) gives an excellent description of the syndrome and related conditions in the shoulder.

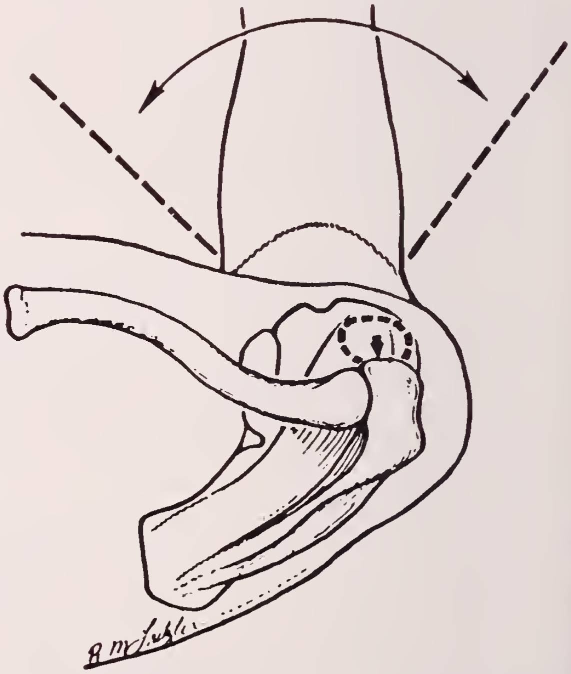

Figure 3. Functional range. Most upper extremity activities are performed with hand in front of shoulder not lateral to it. Impingement occurs against undersurface of anterior third of acromion and occasionally acromioclavicular joint rather than laterally. Wear is centered on supraspinatus. (From Neer, C.S.: Clin. Othop. 173:70, 1983)

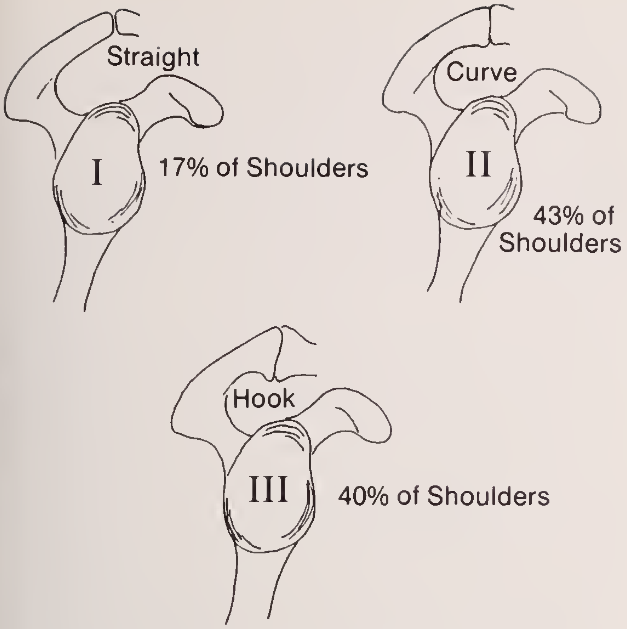

Figure 4. Modified from Bigliani, 1987

The concept of anterior acromioplasty has since evolved as a satisfactory surgical treatment for impingement symptoms. It is selected cases in Stage II and III which most frequently come to surgical treatment after failed conservative care. Dr. Neer clearly described indications for surgery in patients who were good surgical risks and have a reasonable life expectancy. He performed the anterior acromioplasty: first, routinely for rotator cuff repair after a positive arthrogram; second, in patients older than 40 years of age, but with negative arthrograms and with persistent disability after a year of adequate conservative treatment, in whom pain can be temporarily relieved by means of an injection of a local anesthesia into the subacromial space (the impingement test); and third, carefully selected patients younger than 40 years of age with chronic Stage II impingement lesions, who were noted to have a prominent anterior acromion. There is a fourth group of patients undergoing other surgical procedures for conditions in which the rotator cuff and long head of the biceps are weak and ineffective in preventing proximal migration of the head of the humerus.

In 1985, at the First Open Meeting of the American Society of Shoulder and Elbow Surgeons, a small series reporting early success with arthroscopic subacromial decompression was reported by Harvard Ellman (7). It was on the basis of his report, as well as personal observations of open acromioplasty and experience with arthroscopic surgery in other joints which led to the material basis of this report.

Patients

There were 23 shoulders operated in 22 patients, 19 male and three female. Nineteen shoulders were dominant, and four were non-dominant. Indications for surgery were evidence of continuing mechanical pain in the shoulder after failed conservative care as evidenced by pain or limitation of motion in reaching or overhead activities, and a positive impingement sign or test. Usually a patient appeared for treatment after significant interference with life-style or work activities. There was a definite history of injury in 11 of the 22 patients. Of these, eight were workers' compensation (36%). Others noted only a gradual increase in symptoms, reaching a peak by the time they were seen but could not recall any particular precipitating injury.

Average time to surgery from the initial visit was 10.5 weeks with a range of 1–25 weeks. One patient who had a long history of shoulder pain and had shown a poor response to Cortisone injection and physical therapy prior to being seen was operated one week after being seen. He had a painful, stiff shoulder, subacromial bone spurs on x-ray, and a negative arthrogram when first seen. Another patient who was referred after previous treatment including physical therapy and failed surgery for other shoulder conditions was operated three weeks after having been seen.

In all other patients, a course of physical therapy was begun under the supervision of a licensed therapist. The program consisted of range of motion, therapeutic modalities and strengthening of the shoulder abductors and rotators. Patients who responded to physical therapy are not included in the series. Ordinarily this would be a

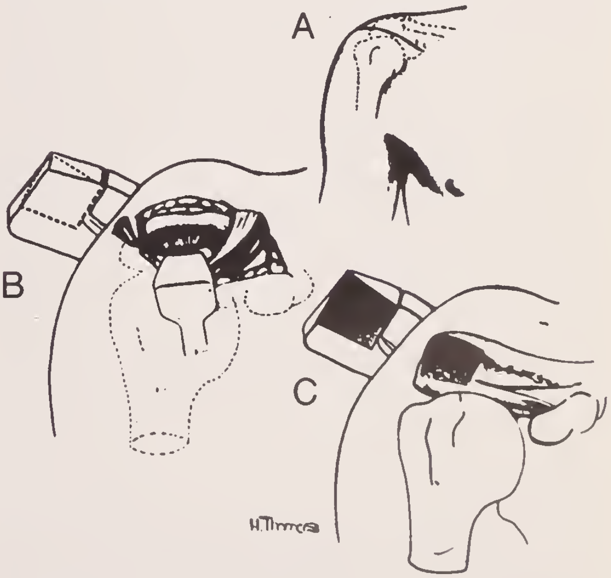

Figure 5. Acromioplasty. A – Superior incision for acromioplasty or rotator cuff repair. B – Outline of bone to be removed from anterior half of acromion. C – In performing acromioplasty one attempts to avoid the acromioclavicular joint. If there are spurs under the joint, these may be smoothed off with a burr. From: Shoulder Surgery in the Athlete, ed. by Jackson

Figure 6

Stage 1: Edema and hemorrhage

Typical age	<25
Differential diagnosis	Subluxation Acromioclavicular joint arthritis
Clinical course	Reversible
Treatment	Conservative

Stage 2: Fibrosis and tendinitis

Typical age	25-40
Differential diagnosis	Frozen shoulder Calcium
Clinical course	Recurrent pain with activity
Treatment	Consider bursectomy or coracoacromial ligament division

Stage 3: Bone spurs and tendon rupture

Typical age	>40
Differential diagnosis	Cervical radiculitis Neoplasm
Clinical course	Progressive disability
Treatment	Anterior acromioplasty Rotator cuff repair

Modified from Neer, C.S., II: Clin. Orthop. 173:70, 1983.

large percentage of patients. But in this series, it was a small number because many patients were referred or referred themselves after already having had some type of prior treatment. There was a significant incidence of associated cervical spine disease (five), and acromioclavicular joint arthritis (12) in the operated series. There were two patients with small rotator cuff tears in the series. A preoperative arthrogram was obtained when there was reason to suspect a rotator cuff tear. Of six arthrograms obtained in the series, only one was positive. A decision to operate was not made as long as the patient was showing any improvement with conservative therapy. In most cases, operation followed only when therapy was not only failing to help but also actually began aggravating the condition.

Methods

All patients were operated in an outpatient surgery setting under general anesthesia (Figure 7). The patient was anesthesized, intubated, and then the shoulder was examined under anesthesia. The patient was transferred into the decubitus position on the operating table and positioned with 7–17 pounds of traction using a shoulder holder. The amount of traction was dependent upon the patient's body size.

The arthroscope was routinely placed through the posterior portal and glenohumeral arthroscopy performed prior to any other surgery. Occasionally, it was necessary to establish an operative portal into this space through the anterior approach to probe or debride. Following this, the instruments were placed in the subacromial space and an additional lateral acromial portal was established for the introduction of the arthroscopic motorized burr. Occasionally, outflow cannulas from the anterior or posterior approach were established to minimize shoulder swelling during the procedure. Lactated Ringers solution was used for irrigation.

Often, it would be necessary to use an arthroscopic cartilage or soft tissue shaver or an arthroscopic currette to remove bursal tissue to improve visibility. In the early part of the series, a rotary suction burr (Zimmer–Hall) was used which was not very aggressive, making the procedure very tedious. The arrival of the (Acufex) notchplasty burr greatly facilitated the anterior acromioplasty part of the procedure. Two marking needles showing the limits of the acromioclavicular joint and the lateral acromion were placed to define the medial and lateral extent of the anterior acromion inside the subacromial space. Then the anterior lateral 1/3 of the acromion was removed with the burr. The coracoacromial ligament was transected using either a retrograde knife or a cutting cautery, or both. Later in the series, a partial resection of the coracoacromial ligament was performed in addition to the transection using a basket rongeur. Hemostasis was accomplished when necessary with an intra–articular electrocautery.

Operative times ranged from one to two hours. The longer operative times were necessary when a Mumford arthroplasty was required in addition to the subacromial decompression procedure. The Mumford procedure was performed open and gave an opportunity to check the completeness of the acromioplasty.

Three patients required admission to a 23–hour observation unit because of post–operative nausea. The other patients were seen in the office for a dressing change the following day. Three patients required interscalene block administered by the anesthesiologist for control of postoperative pain unresponsive to oral narcotics. All patients were discharged ambulatory in an arm–sling and range of motion was begun on the first postoperative day. Codman's pendulum exercises were demonstrated to the patient for use during the first postoperative week. Use of the arm for personal hygiene activities and simple grasping and reaching activities at waist or chest level was encouraged during the first week. At the end of the first week, physical therapy was initiated if the patient was not capable of beginning above shoulder range of motion activities. Patients who were capable of beginning this motion were allowed to do so on their own and went to therapy when strengthening exercise was begun, if they required it. Physical therapy began with range of motion and progressed to resistive exercises for strengthening of the rotators and abductors.

RESULTS

Questionnaires were sent to all of the operated patients with 21 responses. Follow up periods ranged from 4–29 months. Based on questionnaire responses and clinical evaluation there were nine excellent results (39%). These patients had 90–100% relief of their pain and were able to return to full desired activity including sports, work and personal care. There were nine good results (39%) as determined by 70–90% relief of pre-operative pain, the ability to return to work, to accomplish personal care and relief of night pain. Of the remainder, there were four fair results in patients who had improvement in their pre-operative condition but less than 70% relief of their pain and were not able to return to work. There was one poor result in a patient whose pain was not improved. Of the people who were unable to return to work, all were workers' compensation cases.

There were no infections, nerve or vessel injuries. Two cases of heterotopic bone formation were in the acromioclavicular area after Mumford procedure. One did not prevent a good result. The other was in a compensation patient and required excision.

Three patients required re-operation. All of these were workers' compensation patients. One was the second patient in the series, and it was felt at the time of arthroscopic acromioplasty that the decompression might be unsatisfactory for technical reasons when his impingement pain continued. After a reasonable recovery period, it became apparent the patient was now limited by painful arthritis of the AC joint. He was re-operated for the Mumford arthroplasty and the subacromial space was explored. The findings were that the ligament had been sufficiently released but that the anterior acromion required more decompression. He was able to return to work after this operation.

Another patient had excision of heterotopic bone. This patient had a Mumford arthroplasty one year after arthroscopic acromioplasty. Subacromial arthroscopic examination and digital examination of the subacromion at that time had revealed adequate decompression. Heterotopic bone formed after the Mumford arthroplasty procedure. This patient has not returned to work.

The third patient was improved after arthroscopic acromioplasty, but still had symptoms of subacromial impingement. Re-operation was performed open by another surgeon. It appeared to that surgeon that too little bone had been removed. This patient has still not returned to work.

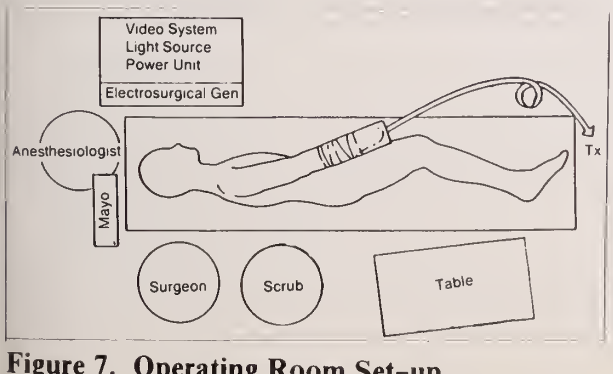

Figure 7. Operating Room Set-up

DISCUSSION

Anterior acromioplasty has become known as a proven procedure in relief of shoulder pain for Impingement Syndrome with related conditions of the subacromial bursa and biceps tendon after conservative treatment has failed. Arthroscopic acromioplasty is a recent development which shows promise in the evolution of treatment for appropriate shoulder conditions where its major advantage is the minimal surgical downtime related to postoperative pain and disability. Hospitalization is usually not required. Patients are able to begin activities of personal hygiene within a few days and often return to work in less than a week. Recovery often is complete at four weeks. Some patients reported partial relief of their impingement pain less than 24 hours after surgery. Failure of anterior acromioplasty is usually not related to inadequate bone or ligament resection but more often is related to failure of diagnosis pre-operatively (8,9,10). In this series, the persons who failed to return to work and resume their normal activities were workers' compensation cases.

The relative ease with which the surgery can be accomplished can bring many patients to treatment far earlier than would normally be the case with open surgery, avoiding the months of disability with a painful, stiff shoulder and progression of the impingement condition to an actual rotator cuff tear.

Pre-operative diagnosis is very important. It should be understood that Impingement Syndrome may be a primary condition in the shoulder, but impingement pain may be secondary to other factors, such as cervical spondylosis, instability of the shoulder, subluxing biceps tendon, and arthritis of the acromioclavicular or glenohumeral joint. Frequently, the impingement condition leads to a painful, stiff shoulder. This can often be confused with an adhesive capsulitis and treated by manipulation under anesthesia and a long course of physical therapy with no improvement until the impingement lesion is resolved. Arthroscopy is not indicated for adhesive capsulitis (11). But many cases of painful, stiff shoulder attributable to the Impingement Syndrome but mistakenly diagnosed as adhesive capsulitis can achieve dramatic relief with this surgery. In these cases, manipulation under anesthesia can accompany the procedure and should precede the arthroscopic procedure.

The percentage of good or excellent results (78%) in this series is less than Ellman's series (88%). His report used a similar, but more objective means of evaluating results, and had fewer work-related injuries (24% vs 39%). If only noncompensation injuries are evaluated, there were 100% good or excellent results (50% excellent, 50% good), but the series becomes too small to be relevant. It appears that better pre-operative diagnosis could have led to improved results. In retrospect, two of the patients in the fair results category probably had mild anterior instability mistakenly diagnosed as primary impingement. Ellman describes this problem as being an important differential (12).

Most of the shoulders in this series were improved to a significant degree. The failure of a portion to return to normal activities is seen as a failure of the workers' compensation system, not of the arthroscopic acromioplasty as a procedure. The results in noncompensation patients as a whole were impressive. A few of these patients, in spite of postoperative pain, returned to the office the day after surgery enjoying relief from their impingement pain. The disparity of results in the group of workers' compensation patients is similar to that seen in this group after surgical treatment of other conditions.

The advantages of arthroscopic acromioplasty are in the relative ease with which the surgery can be accomplished and the short recovery period. This is because of the small incisions used and because the deltoid muscle origin is not removed to accomplish the surgery. The result is that the patient can begin range of motion activities as soon as pain allows, usually in a few days and always within a week. The long term results should not be significantly different from open anterior acromioplasty, as described by Dr. Neer.

The procedure is technically demanding for the arthroscopic surgeon. Like arthroscopy of the knee, it has the advantage of offering less traumatic treatment than open methods. The presence of a small rotator cuff tear does not preclude athroscopic treatment. Based on this series, arthroscopic treatment can achieve a good result in patients with a tear smaller than one centimeter. Because the goal of treating rotator cuff lesions many times is pain relief, arthroscopic treatment might even be preferred for various reasons in some cases (i.e., time off work, hospital stay, cosmesis). A longer recovery period after surgery in these patients should be expected. Although it has been used by some surgeons, this author does not recommend arthroscopic acromioplasty for Stage III impingement with a rotator cuff tear larger than one centimeter. In suitable candidates it is preferable to repair these injuries by open means. The use of a preoperative arthrogram is always helpful in making this decision. Arthroscopic treatment, by nature of its opportunity to examine a joint from a different perspective with limited side effects, provides another way to evaluate and treat the shoulder joint, although it may not always be preferable to traditional treatment methods.

Author's note: Since this paper was written one of the patients in the "fair" category has returned to work, bringing his result into the "good" category.

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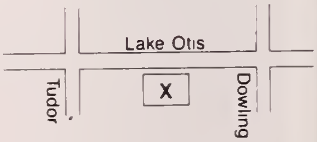

History of Medicine in Alaska

ROBERT BOCOCK WILKINS, M.D.

Lynchburg, in central Virginia, was old and conservative. At the time Bob Wilkins grew up there, it had a population of about 40,000 people and rejected the building of Southern Railroad shops because it preferred being a white collar town. In fact, it was the home of several colleges, among them Randolph Macon Women's College and Sweet Briar.

It was a pleasant, if uneventful, place to live. For a young boy, life was centered on the cliffs and in the woods above and around the James River.

As a child Bob fleetingly saw Gil Blankinship who also was born in Lynchburg. Gil would visit his grandmother, a neighbor of the Wilkins, on Sunday afternoon. While a few years younger than Bob, he made an impression in his sailor suit complete with whistle.

Bob Wilkins attended the University of Virginia at Charlottesville. This must be one of the United States' most beautiful campuses. It was designed by Thomas Jefferson who was an architect of considerable skill and who ranked his founding of the university much before having been president.

For Bob, who perceived himself at this time as an unsophisticated boy from a small town, it was a gradual introduction to people from many parts of the country. However, without the $120 lent to him by the Kiwanis, he would not have been able to attend. The university tuition plus room and board was $595 his first year. He was able to stay in school because of jobs through the National Youth Administration, one of Franklin Roosevelt's programs to assist in the depression, and through scholarships such as the one granted by the Dupont Foundation.

Although he majored in biology, Bob was not sure while in college that medicine was his choice. However, he stayed in a boarding house frequented by medical students which may have piqued his interest. He was graduated Phi Beta Kappa from college and later Alpha Omega Alpha from the University of Virginia Medical School.

During the latter, he was in the V–12 program which the Navy had established during the 1940s to provide physicians for war time needs. Unfortunately for Bob, he joined early during a time the Navy still felt its midshipmen were equivalent to its apprentice seaman and should learn how to wash their white uniforms by hand.

Bob knew he wanted to leave Virginia to see the world. Internship provided the opportunity.

Bob's maternal grandparents had lived in Panama. His grandfather, an engineer, worked for the government during the construction of the canal. The Alaska Railroad was built with Panama Canal equipment that still had good use in it. His grandmother worked as a foreign correspondent writing articles about Panama. Eventually, they were to retire to the San Juan Islands in Washington. Because Bob had visited them when he was 12, he was somewhat familiar with the Northwest.

He applied to and was accepted by the Virginia Mason Clinic, established by Dr. Tate Mason who, after World War I, moved from Virginia to Seattle. The clinic encouraged Virginians to come for training, and Bob was one of these. He was to serve a 15 month rotating internship. After World War II, it became necessary to reestablish a regular 12 month schedule. During the war, there had been nine month internships.

Afterwards he spent two years on active duty in the Navy as a lieutenant, j.g., to meet the obligations of the V–12 program. He was first stationed at Bremerton where he went to sea on a destroyer. The ship got as far as the Straits of Juan de Fuca before its boiler broke. Thus ended Bob Wilkins' sea duty. For the remainder of his service, he was stationed at the Office of Naval Officer Procurement in Seattle.

Then he returned to Virginia Mason Hospital to serve two years in medical residency. These were split by a year spent in physiology. At the time, it was thought that in addition to clinical work, good training was to be had in intensive review of basic science. Bob received a Master of Sciences from the University of Washington in 1951.

Because there were so many patients from Alaska in the Virginia Mason Hospital, Bob had a splendid introduction to Alaska with tales of varying degrees of veracity but no quibble over the enticing spirit of Alaska.

Bob was recruited by the Anchorage Medical and Surgical Clinic for a summer. He flew to Anchorage in a stratocruiser which was a large plane to him. When he arrived, he found a few blocks of 4th and 5th Avenues paved, and the Seward Highway was completed and opened later that summer. Howard Romig flew him to Cooper's Landing, the only way to get there at that time.

The Anchorage Medical and Surgical Clinic was located in the Lingo House at 330 L Street. Bob was to live in one of the apartments above the offices. The clinic was comprised of Howard Romig, Charles St. John, Asa Martin, and Jack Sedwick.

When it became clear that Bob Wilkins was in Anchorage to stay, he moved to the Knik Arms. Anchorage was such a relatively new community that many things were needed. There were infinite opportunities for the doer, and that Bob was. It was the challenge he had sought in contrast to his staid and conventional upbringing.

The Greater Anchorage Health District, part of the

Territorial Health Department, was to catch his attention. Bob was to serve first as acting health officer as a volunteer and then for 10 years as a member of its board until well into statehood. Bob stayed until 1971 on the Greater Anchorage Borough Board of Health, serving as president for six years.

Bob Wilkins was instrumental in starting the Anchorage Medical Society. The physicians in private practice in Anchorage met with the doctors on the Elmendorf Air Force Base but were overwhelmed by the number of military compared to the locals. In particular, there was no opportunity to discuss medical business and the problems engendered by private practice. Bob chaired the founding committee and served as the Society's president in 1954.

In addition, he became the indispensable executive for the Alaska State Medical Association, serving as secretary-treasurer from 1954-67 and 1969-71, and as president, 1967-69. At one time Bob knew every doctor in the state. When he first came, there were eighteen doctors on the scene. When the census grew to 150, paid staff took over.

Bob also was a founding member and later president of the Alaska Heart Association and served as a national delegate to the American Heart Association. These were all needs to be met, and it was a lively business to be able to provide a solution.

The outstanding event of Bob's early years in Anchorage was the polio epidemic of 1954. He recalls there were ten deaths from bulbar polio and approximately 100 cases of paralytic polio. Many kinds of respiratory devices were used, and it was necessary to import help from Seattle: both equipment and physicians. Bob is under the impression that considering the size of the Anchorage population, this was a bad outbreak. The polio epidemic is recalled vividly by all physicians in Anchorage at the time.

In 1955 Bob Wilkins and Marilyn Elizabeth North were married. She was a graduate of Northwestern University and had done postgraduate studies in art at Claremont College and the University of Mexico. Hired as a school teacher, she and her brother drove the Alaska highway. Later, she became art supervisor for the Anchorage School District, and subsequently she was elected to the school board, serving for six years.

Their first born, Roberta Marie, has the distinction of celebrating her eighth birthday this year, 1988. She is one of those puzzling Leap Day babies, born on the 29th of February, 1956. The next year, on March 10th, Lee Murray was born.

Because music meant much to both Bob and Marilyn, different opportunities emerged. Particularly, they were eager to provide a stimulating environment for their children. Lee has become a professional violinist and returns yearly to Anchorage as concert master of the Basically Bach Festival. He is busy in New York with the Hampshire String Quartet.

As early as 1956, Bob began his association with the Anchorage Concert Association which has brought to Anchorage outstanding performers in music and dance. Bob Wilkins has served as managing director from 1960 to the present. Through the years the concert association has grown, increasing the number of performances while retaining the quality of the performances. The Concert Association has been a major contributor to the cultural life of the Anchorage community, in no small part because of the determination and hard work of Bob Wilkins. He admits to having enjoyed meeting so many fabulously talented people through the years, a small reward for diligent labor.

When he was first in Anchorage, he remembers how primitive things were. The hospital facilities, for example, were not the same quality as those run by the Sisters of Providence in Seattle. There was a frontier spirit to all things. EKGs had to be put on photographic paper developed in a dark room. There was no radiology. Bob did his own GI fluoroscopy.

He traveled with Milo Fritz to Tyonek where he did the physicals, and Milo did the eye, ear, nose and throat examinations. Bob admires Milo Fritz for his determination to do something about the horrendous problems, especially with ear disease, that occurred in the bush during the 1950s. It was a one man effort to get the Public Health Service to acknowledge the problem and eventually do something about it. In the meantime, Milo tackled the problem.

The earthquake of 1964 is a memorable event in Anchorage. It was not a major medical emergency, fortunately. However, because there was a gas leak in the area of the Presbyterian Hospital, all the patients were evacuated to relatively untouched Providence Hospital. The only major problem was that the elevators did not function. Bob remembers carrying patients upstairs.

Bob Wilkins has enjoyed the people and personalities he has encountered through the practice of medicine. He has found it a great experience. He still maintains a busy office practice and hopes to continue to do so.

The Alaska State Medical Association has honored him both as a "Physician of the Year" and with the A. H. Robins "Community Service Award."

Bob Wilkins quoted legendary physiologist A.J. Carlson as saying, "A good canary will sing in any cage." A good physician and activist such as Bob will perform in any situation.

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Glimpses of Alaskan Medical History

Edited by Robert Fortuine, M.D.

Smallpox decimates the Tlingit (1787)

The English trader Captain Nathaniel Portlock sailed his vessel the *King George* to the northwest coast for the second time in 1787. After several weeks in Prince William Sound he headed southeastward, ultimately finding a haven in what he called Portlock Harbor, located on the western shore of Chicagof Island about 30 miles southeast of the mouth of Cross Sound. There he traded with the Indians for a couple of weeks, while sending an exploring party by longboat as far south as Norfolk Sound.

The text presented is from Portlock's own account (1) of his voyage and was written in August 1787, at the time his vessel was anchored at Portlock Harbor. In it he describes several local Indians with the characteristic scars of smallpox and speculates that the disease was introduced by the Spanish in 1775. It is true that a tiny Spanish vessel under the command of Lt. Juan Francisco de la Bodega y Quadra had reached an area somewhat north of Norfolk Sound in 1775. Four years later two other Spanish ships, under Ignacio Arteaga and Bodega, respectively, sailed along the Alaskan north Pacific rim, making an extended visit to Bucareli Bay (near presentday Craig), with shorter stays on Hinchinbrook Island and Elizabeth Island (Cook's Cape Elizabeth).

The diaries from these two voyages do not mention smallpox (the symptoms of which were known to everyone), although a number of men from the voyage of 1779 were reported to have been afflicted while at Bucareli Bay by some type of infectious disease. There is no mention of the disease having been spread to the Natives.

No other European ships are known to have passed this way except those of Cook, who did not land in southeastern Alaska. Despite Portlock's obvious enthusiasm for laying the blame on Spain, it is more probable that smallpox was introduced to the Tlingit by neighboring Indian tribes from the south and east.

"I observed the oldest of the men to be very much marked with the small-pox, as was a girl who appeared to be about fourteen years old. The old man tried to describe the excessive torments he endured whilst he was afflicted with the disorder that had marked his face, and gave me to understand that it had happened some years ago. This convinced me that they had had the small-pox among them at some distant period. He told me that the distemper carried off great numbers of the inhabitants, and that he himself had lost ten children by it; he had ten strokes tatooed on one of his arms, which I understood were marks for the number of children he had lost. I did not observe any of the children under ten or twelve years of age that were marked; therefore I have great reason to suppose that the disorder raged a little more than that number of years ago; and as the Spaniards were on this part of the coast in 1775, it is very probable that from them these poor wretches caught this fatal infection. They, it should seem, are a nation designed by Providence to be a scourge to every tribe of Indians they come near, by one means or another.

"The Spaniards were among them in the height of the summer, and probably they caught the infection about the month of August. To see their manner of living at that season of the year, one would think it a miracle that any of them escaped with their lives. I found men, women, and children, all huddled together in a close house near a large fire, and entirely surrounded with stinking fish. Round the house for at least one hundred yards, and all along the banks of a little creek that ran down by this miserable dwelling, were strewed stinking fish; and in several places were beds of maggots a foot deep, and ten or twelve feet in circumference:... Possibly the small-pox only raged during the warm weather, and the infection was destroyed by the setting in of a severe winter; but the sufferings of the poor Indians, when the disorder was at its height, must have been inconceivable, and no doubt the country was nearly depopulated; for to this day it remains very thinly inhabited.

"A number of the Indians who visited us from the Eastward were marked with the small-pox, and one man who had lost an eye gave me to understand that he lost it by that disorder; but none of the natives from the Westward had the least traces of it. I cannot account for this circumstance any other way than by supposing that the vessel from which these unfortunate people caught the infection, was in a harbor somewhere about Cape Edgecombe [sic]; and perhaps none of the natives further to the Westward than this Sound had an opportunity of having any intercourse with her, and by that means happily escaped the disorder".

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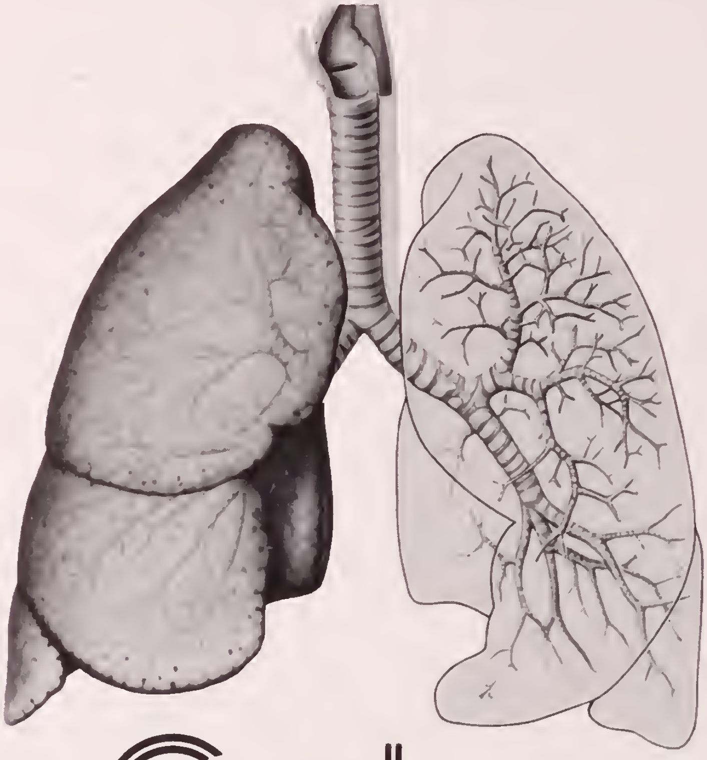
Consider the causative organisms...



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- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN o serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose wit Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucos enzymatic test strip, Lilly). [061787L

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President's Page

Dear Fellow Alaskan Physicians,

This will be my last column to you as president of ASMA. Dr. Irvin Rothrock will take over as president at our annual meeting in Fairbanks, June 8-11. Needless to say, we hope to see you there along with your family. To have served as President of the Alaska State Medical Association has been a rather interesting and worthwhile experience, to say the least. There have been several minor problems that have come up, and one or two rather large ones. Hopefully, these have been settled to the benefit of all parties concerned. I must say that the duties of this office would have been a lot more stressful, certainly a lot more strenuous, and much more time consuming if it had not been for the efforts of Ray Schalow, our Executive Director, Gayle Mathiesen, and all the other members of the staff. I appreciate their time, loyalty, and efforts on behalf of ASMA.

As you know by now, SB211, the Tort Reform, bill did not get out of the House Judiciary Committee, thanks to the efforts of Representative Sund and the three other attorneys that were members of that committee. You must admit, however, that our legislative members certainly had the health of their constituencies at heart since they passed the chiropractic bill SB264 which expands the chiropractic scope of medicine. They are now able to do school exams, sports exams, workers compensation physicals, etc.. It's rather disheartening when the legislature passes a bill such as this and will not act upon significant tort reform.

I would be remiss if I didn't extend our thanks and appreciation to Dave McGuire for his efforts to obtain meaningful tort reform in this past session. It never ceases to amaze me when I consider the time, effort, and costs to Dave in his repeated speeches, his efforts on behalf of Citizens Coalition for Tort Reform, and his many trips to Juneau for lobbying — all in an effort to obtain passage of SB211 this spring. He stayed to the bitter end of the legislative session, and because of his efforts the votes were there to pass SB211 had it come to the floor. There was rather significant maneuvering during the last two or three days of the legislative session. The combined Senate/House Bill 85 almost made it to the floor where it would have passed, and we would have had meaningful tort reform. Thank you, David, for your efforts. We certainly hope you will continue your work.

Dr. Irvin Rothrock, convention committee chairman, and Dr. Charles Steiner, CME committee chairman of the annual meeting, have put together an exceptionally fine program. We look forward to seeing you in Fairbanks.

One last plea before I close. I would urge all of you as members of the medical community in this state to become involved in the political process. It's the only way we will ever obtain significant tort reform.

Thank you again for your membership, time, thoughts and service to ASMA in the past year.

C. Jerry Little, M.D.
President
Alaska State Medical Association

Rasmuson Renamed to Panel

Elmer Rasmuson, a former Anchorage mayor, has been reappointed to the Arctic Research Commission. President Reagan reappointed Rasmuson to a second term on the commission created to develop an Arctic research policy and programs. Rasmuson, chairman of the National Bank of Alaska executive committee, received his first appointment in February 1985. Other Alaskans on the research commission are Juan Roederer of Fairbanks and Oliver Leavitt of Barrow.

Executive Privilege

Accountability to the public through assurance of competent care to patients by physicians and other health professionals is a paramount responsibility of organized medicine. Occasionally such accountability is jeopardized by physicians whose functioning has been impaired by psychiatric disorders, including alcoholism and drug dependence. An equally important issue is the effective treatment and rehabilitation of the physician patient so that he can be restored to a useful life.

It is a physician's ethical responsibility to take cognizance of a colleague's inability to practice medicine adequately by reason of physical or mental illness, including alcoholism or drug dependence. Ideally, the affected physician himself should seek help when difficulties arise. Often, however, he is unable or unwilling to recognize that a problem exists. When exhortations by family and friends are ineffective and when the physician is unable to make a rational assessment of his ability to function professionally, it becomes the responsibility of colleagues to make that assessment for him, and to advise him whether he should obtain treatment and curtail or suspend his practice.

In almost all cases of physician impairment, there is a family sharing the problems that become manifest. While an impaired physician is seeking treatment, it is extremely important that the family be able to do the same. Emotional support is tremendously important and beneficial for the impaired physician's family during this time.

The American Medical Association has for a long time maintained the position that chemical dependency should be treated as an illness and that the patient is entitled to the same legal rights and medical treatment opportunities accorded patients with other illnesses. The AMA's Council on Ethical and Judicial Affairs submitted the following two opinions to the House of Delegates at the December 1987 Interim Meeting.

- In order to protect patients and the public, physicians are required to report to the appropriate body credible evidence of impairment that affects competence in their colleagues. Such impairment may result from abuse of drugs or alcohol, or from mental or physical illness. All physicians have an obligation to urge impaired colleagues to seek treatment.
- It is unethical for a physician to practice medicine while under the influence of a controlled substance, alcohol, or other chemical agents that impair his or her ability to practice medicine.

The Alaska State Medical Association in partnership with the Alaska State Medical Board has developed an Impaired Physicians program that is modeled after the successful Oregon State Medical Association program. A statewide monitored treatment program will provide a fully confidential resource for impaired physicians. In addition, the State Medical Board is firmly committed to referring chemically dependent licensees to ASMA for intervention by the Impaired Physicians Committee. We encourage each of you to contact a member of the Impaired Physicians Committee or myself if you have a colleague that needs support and a path to the program.

We are also intensely concerned with spouse impairment. We hope this program can be offered as a resource to spouse impairment and support, and we will attempt to get that message across in the coming months.

The passage of HB70 this past session created a new Alaska Statute, AS 08.64.336, which states in part; "A physician, hospital, or hospital committee that in good faith submits a report under this section or participates in an investigation or judicial proceeding related to a report submitted under this section is immune from civil or criminal liability for the submission or participation." This statute addresses the fear of lawsuits by those that report impairment in good faith.

My endeavor in writing this article is to develop greater visibility among those who may feel a need for referral but who find a lack of resources when a need exists. Within the next three months, a brochure will be developed that will outline the program and the resources available. We hope to have a telephone number available to respond to any inquiry or crisis within a month or two.

The thrust of our program is to provide a very confidential and rehabilitative approach, rather than one that is punitive and without recourse. The Department of Occupational Licensing and the State Medical Board support this approach and have pledged their support.

Finally, in closing, I ask that each of you thank Dr. Janice Kastella for the long hours, quiet dedication, and support of this program over the past several years. Her previous work in this program saved us many hours of time and tribulation. Her care and concern for her colleagues provided the impetus for this program. Thank you, Doctor.

Raymond Shalow
Executive Director
Alaska State Medical Association

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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
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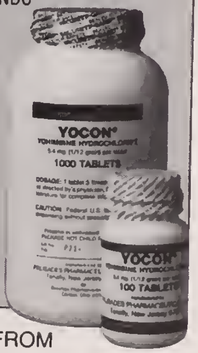
Rev. 1/85



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INDICATIONS AND USAGE: BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1. For therapeutic use in patients with hypokalemia with or without metabolic alkalosis, in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.

2. For the prevention of potassium depletion when the dietary intake is inadequate in the following conditions: Patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis with ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy, and with certain diarrheal states.

3. The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated

CONTRAINDICATIONS: Potassium supplements are contraindicated in patients with hyperkalemia since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: Chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g , spironolactone, triamterene).

Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium.

All solid dosage forms of potassium chloride supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation.

WARNINGS: Hyperkalemia—In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Interaction with Potassium Sparing Diuretics—Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g , spironolactone or triamterene) since the simultaneous administration of these agents can produce severe hyperkalemia

Gastrointestinal Lesions—Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage or perforation.

K-DUR tablets contain micro-crystalloids which disperse upon disintegration of the tablet. These micro-crystalloids are formulated to provide a controlled release of potassium chloride. The dispersibility of the micro-crystalloids and the controlled release of ions from them are intended to minimize the possibility of a high local concentration near the gastrointestinal mucosa and the ability of the KCl to cause stenosis or ulceration. Other means of accomplishing this (e.g., incorporation of potassium chloride into a wax matrix) have reduced the frequency of such lesions to less than one per 100,000 patient years (compared to 40–50 per 100,000 patient years with enteric-coated potassium chloride) but have not eliminated them. The frequency of GI lesions with K-DUR tablets is, at present, unknown. K-DUR tablets should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis—Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, potassium acetate, or potassium gluconate.

PRECAUTIONS: The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis per se can produce hypokalemia in the absence of a deficit in total body potassium while acute acidosis per se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease, or acidosis requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the electrocardiogram, and the clinical status of the patient.

Laboratory Tests: Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis.

Drug Interactions: Potassium-sparing diuretics; see **WARNINGS.**

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term carcinogenicity studies in animals have not been performed.

Pregnancy Category C: Animal reproduction studies have not been conducted with K-DUR. It is also not known whether K-DUR can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. K-DUR should be given to a pregnant woman only if clearly needed.

Nursing Mothers: The normal potassium ion content of human milk is about 13 mEq per liter. Since oral potassium becomes part of the body potassium pool, so long as body potassium is not excessive, the contribution of potassium chloride supplementation should have little or no effect on the level in human milk.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS,** and **OVERDOSAGE).** There have also been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS);** other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea. These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely

OVERDOSAGE: The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic electrocardiographic changes (peaking of T-waves, loss of P-waves, depression of S-T segment, and prolongation of the QT-interval). Late manifestations include muscle-paralysis and cardiovascular collapse from cardiac arrest.

Treatment measures for hyperkalemia include the following:

1. Elimination of foods and medications containing potassium and of potassium-sparing diuretics.
2. Intravenous administration of 300 to 500 ml/hr of 10% dextrose solution containing 10–20 units of insulin per 1,000 ml.
3. Correction of acidosis, if present, with intravenous sodium bicarbonate.
4. Use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia, it should be recalled that in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity.

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BRIEF SUMMARY

CONTRAINDICATIONS

There are no known contraindications to the use of sucralfate

PRECAUTIONS

Duodenal ulcer is a chronic, recurrent disease While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract The clinical significance of these animal studies is yet to be defined However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted

Pregnancy: Teratogenic effects Pregnancy Category B Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS

Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4 7%)

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo

OVERDOSAGE

There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION

The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be take within one-half hour before or after sucralfate

While healing with sucralfate may occur during the first week or two, treatmen should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray o endoscopic examination

HOW SUPPLIED

CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088-1712-47 and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49). Light pink score oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by C's o the other

Issued 1/8

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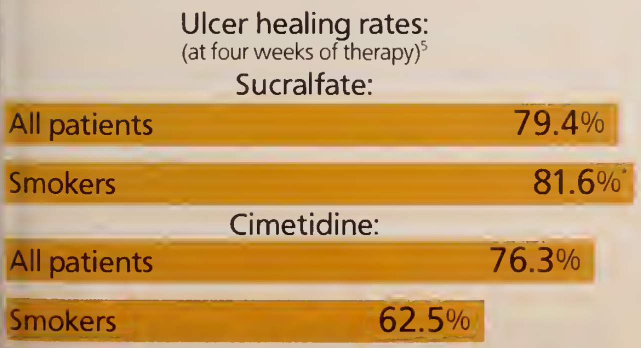

*Significantly greater than cimetidine smoker group ($P < .05$).

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0825A8

ARMY RESERVE MEDICAL PROFILE NO.7

dier being examined for effects of high-altitude cerebral edema.

ALLAN J. HAMILTON, M.D.

Neurosurgical Resident and Research Fellow, Massachusetts General Hospital, Boston, Massachusetts. Captain, U.S. Army Reserve.

EDUCATION Ithaca College, B.A. (Magna Cum Laude) Hamilton College (Pre-med); Harvard Medical School.

RESIDENCY General Surgical Internship. Neurosurgical Residency, Massachusetts General Hospital.

CONTINUING EDUCATION Neurology and Neuro surgery Research Fellowship Training, National Institutes of Health.

OUTSTANDING ACHIEVEMENTS Olsen Memori Fellowship, National Masonic Medical Research Foundatio Albert Schweitzer Fellowship, International Albert Schweitz Foundation; Harvard Medical School Cabot Prize for Best Senior Thesis; recently published article, "Who Shall Live and Who Shall Die" in Newsweek Magazine.

"The work I'm doing in the Army Reserve fits perfectly with my academic research interests in civilia life. The Army is very concerned with the effects of high-altitude cerebral edema, which is a mirror mode of cerebral hypoxia, something I deal with every day in our neurosurgical intensive care unit. I couldn't ask for a smoother transition. And that's true for a lot of Reserve physicians. All we really do is change our clothe not our mindset.

"Some of the projects the Army is undertaking are on the cutting edge of research. For example, I'm currently involved in developing for the Army a proto type of a non-invasive intracranial pressure-monitorin device that we hope will allow us to measure pressure changes as the brain swells—without drilling holes in the skull. If we can get our design to work, such a device could revolutionize high-altitude medicine as w as civilian neurosurgical care.

"The quality of medicine and the caliber of peop I've been associated with in the Army Reserve are, without question, equal to civilian hospitals. In fact, I giving serious consideration to applying for an active duty academic position in Army Medicine when my residency ends at Massachusetts General."

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Auxiliary News

Alaska State Medical Association

I would like to start off with a hearty salute to one of our own! Auxilian **Agnes Coyle** received a **First Lady Volunteer Award** for her efforts in the Alaska Native Center gift shop. This award honors the cream of the crop among volunteers and Agnes certainly deserves it! Nice going, Agnes!!

Many of our spouses are uncomfortable receiving compensation for their work as expert witnesses in malpractice trials. I would like to announce an entirely **voluntary** new program. Henceforth, the Alaska State Medical Society is prepared to receive the compensation checks with the proceeds to be used for tort reform efforts. You will be hearing more about this. Remember, it is **voluntary**.

In my last column, I wrote about our first efforts in grass roots politics. Many of you know that our tort reform resolution was accepted in four districts throughout the state in the recent Republican conventions. This means that tort reform will be debated at a state level. If the resolution passes the state and is adopted as a platform of the Republican Party, it immediately impacts any Republican legislator who is anti–tort reform. The party make it very uncomfortable for legislators who do not follow the party platform. Had we known this two years ago, the four Republican legislators who voted against tort reform would have had a very difficult time doing so. We would have had tort reform in Alaska.

We are in the process of completing a comprehensive plan for medical participation in grass roots politics. Please get involved. As we called doctors and spouses to attend precinct meetings, we were told several times that they were too busy and didn't want to get involved. We cannot afford not to get involved. In most instances, your involvement will be attending one or two meetings. It is essential that we all participate. We are the second largest employer group in Alaska. If we are organized and everyone does his/her part, we will be able to address the medical concerns of the state.

I would like to close with one statement: Keep talking about tort reform. At our district convention, tort reform was debated in resolutions committee and would have passed unanimously but for one lone dissentee who said he could not vote for any tort reform bill that did not cap attorneys' fees. A doctor had told him that was necessary. Recently, I had some printing done of tort reform material. The owner of the shop told me "Tort reform, you bet. I wrote a letter to my legislator. My doctor told me about it." I think we can win.

Cassie Hill
President, ASMA Auxiliary

Providence Hospital is recruiting for a board-certified physiatrist to serve as Rehabilitation Medical Director for both the hospital and the Alaska Treatment Center.

Providence Hospital is a 303 bed medical center in Anchorage, Alaska's largest city. Rehabilitation services are delivered to orthopedic, pediatric, oncology, burn, cardiac, and neonatal departments from our acute therapy department. In addition, our 20 bed Rehabilitation Center contains its own PT, OT, and Speech-Language services.

The Alaska Treatment Center, comprehensive outpatient rehabilitation facility, is CARF accredited. Programs of care include pediatric, back and ortho/neuro programs, as well as audiology services, driver training, and therapeutic pool.

The Medical Director will work closely with the rehabilitation directors at each facility in areas such as program development, medical staff relations, and continuing education for staff. A part-time private practice is also available.

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Editorial

Arctic science received a major boost when the Alaska legislature established the Alaska Science and Technology Foundation, one of Governor Cowper's highest legislative priorities. The foundation will be funded initially by a $6 million grant, and the legislation authorizes the Commissioner of Revenue to allocate up to $33 million a year in additional funds up to a total endowment of $100 million.

The purpose of the foundation is to stimulate arctic research and enable Alaska to become a leader in arctic science. The foundation will be in the hands of a nine member board and will have a permanent staff that includes an executive director and a few support staff.

Now that the foundation has been created and funds appropriated, the major challenges lies ahead. It will be all to easy for the foundation to fritter away its funds on worthless projects, frivolous science, and incompetent scientists. Already there have been calls for the foundation to fund research "...with immediate payoff for existing industries, not just long-term benefits to science."

Critical to the success of the Alaska Science and Technology Foundation and its future place in Alaska's history will be the quality of its board and the strategies they select to accomplish the goals of the foundation.

During the past five years, Alaskans have devoted incredible energy to establishing a national arctic science policy and agenda. The Arctic Research and Policy Act of 1984, passed by the U.S. Congress and signed into law by President Reagan, provided for the first time ever a national science policy for the arctic. Since the law was enacted, the National Academy of Sciences Polar Research Board, the Interagency Arctic Research Policy Committee, and the U.S. Arctic Research Commission have been actively developing a national research agenda to implement the goals of this landmark legislation. The health community has been in the forefront of this effort. A five-year plan now exists—a product of five years' work, public hearings, input from the scientific community, and several drafts. Nationally, major efforts are underway to implement its recommendations.

Alaskans would do well to examine carefully the five-year plan. Fortunately, the Alaska Science and Engineering Advisory Commission has been actively involved in the plan's development and is to play a major role in establishing priorities for Alaskan science.

But, at this time, it is premature for the Alaska Science and Technology Foundation simply to hand out grants for research. The concept of an endowment is "in perpetuity." In order for the foundation to meet this lofty and excellent goal, *it must ensure a solid foundation for arctic science.*

Numerous impediments to arctic science have been exhaustively documented and eloquently described by researchers and scientists. Arctic logistics; difficulties in obtaining, storing, and sharing data; education and development of future arctic scientists; and administrative barriers to seeking and obtaining research funds all need to be addressed and resolved. These contributions are essential if the foundation is to leave a mark on Alaska's history and be worthy of an endowment "in perpetuity." By creating a climate and infrastructure that enhances opportunities for science, the foundation can make a permanent difference.

Finally, although it should go without saying, I will close by stating that if the foundation directly funds research, then the research should be: 1) open to all scientists to compete for funding, 2) peer-reviewed, 3) of benefit to Alaskans, 4) able to be accomplished, and 5) done with evidence of interagency and inter-organization cooperation and support.

We applaud the Governor for his vision and leadership and our legislature for responding to the challenge and opportunity. But now let us all work together to make the Alaska Science and Technology Foundation a success.

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INDICATIONS AND USAGE

Cipro® is indicated for the treatment of infections caused by susceptible strains of the designated microorganisms in the conditions listed below

Lower Respiratory Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Proteus mirabilis, Pseudomonas aeruginosa, Haemophilus influenzae, Haemophilus parainfluenzae,* and *Streptococcus pneumoniae*

Skin and Skin Structure Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Proteus mirabilis, Proteus vulgaris, Providencia stuartii, Morganella morganii, Citrobacter freundii, Pseudomonas aeruginosa, Staphylococcus aureus* (penicillinase and nonpenicillinase-producing strains), *Staphylococcus epidermidis,* and *Streptococcus pyogenes*

Bone and Joint Infections caused by *Enterobacter cloacae, Serratia marcescens,* and *Pseudomonas aeruginosa*

Urinary Tract Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Serratia marcescens, Proteus mirabilis, Providencia rettgeri, Morganella morganii, Citrobacter diversus, Citrobacter freundii, Pseudomonas aeruginosa, Staphylococcus epidermidis,* and *Streptococcus faecalis*

Infectious Diarrhea caused by *Escherichia coli* (enterotoxigenic strains), *Campylobacter jejuni, Shigella flexneri,** and *Shigella sonnei** when antibacterial therapy is indicated

*Efficacy for this organism in this organ system was studied in fewer than 10 infections

Appropriate culture and susceptibility tests should be performed before treatment in order to isolate and identify organisms causing infection and to determine their susceptibility to ciprofloxacin Therapy with Cipro® may be initiated before results of these tests are known, once results become available appropriate therapy should be continued. As with other drugs, some strains of *Pseudomonas aeruginosa* may develop resistance fairly rapidly during treatment with ciprofloxacin. Culture and susceptibility testing performed periodically during therapy will provide information not only on the therapeutic effect of the antimicrobial agent but also on the possible emergence of bacterial resistance

CONTRAINDICATIONS

A history of hypersensitivity to ciprofloxacin is a contraindication to its use A history of hypersensitivity to other quinolones may also contraindicate the use of ciprofloxacin

WARNINGS

CIPROFLOXACIN SHOULD NOT BE USED IN CHILDREN OR PREGNANT WOMEN The oral administration of ciprofloxacin caused lameness in immature dogs Histopathological examination of the weight-bearing joints of these dogs revealed permanent lesions of the cartilage Related drugs such as nalidixic acid, cinoxacin, and norfloxacin also produced erosions of cartilage of weight-bearing joints and other signs of arthropathy in immature animals of various species (SEE ANIMAL PHARMACOLOGY SECTION IN FULL PRESCRIBING INFORMATION)

PRECAUTIONS

General.

As with other quinolones, ciprofloxacin may cause central nervous system (CNS) stimulation, which may lead to tremor, restlessness, lightheadedness, confusion, and very rarely to hallucinations or convulsive seizures Therefore, ciprofloxacin should be used with caution in patients with known or suspected CNS disorders, such as severe cerebral arteriosclerosis or epilepsy, or other factors which predispose to seizures (SEE ADVERSE REACTIONS)

Crystals of ciprofloxacin have been observed rarely in the urine of human subjects but more frequently in the urine of laboratory animals Crystalluria related to ciprofloxacin has been reported only rarely in man, because human urine is usually acidic Patients receiving ciprofloxacin should be well hydrated, and alkalinity of the urine should be avoided The recommended daily dose should not be exceeded Alteration of the dosage regimen is necessary for patients with impairment of renal function (SEE DOSAGE AND ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION)

Drug Interactions

Concurrent administration of ciprofloxacin with theophylline may lead to elevated plasma concentrations of theophylline and prolongation of its elimination half-life This may result in increased risk of theophylline-related adverse reactions. If concomitant use cannot be avoided, plasma levels of theophylline should be monitored and dosage adjustments made as appropriate.

Antacids containing magnesium hydroxide or aluminum hydroxide may interfere with the absorption of ciprofloxacin, resulting in serum and urine levels lower than desired, concurrent administration of these agents with ciprofloxacin should be avoided

Probenecid interferes with the renal tubular secretion of ciprofloxacin and produces an increase in the level of ciprofloxacin in the serum This should be considered if patients are receiving both drugs concomitantly

As with other broad-spectrum antibiotics, prolonged use of ciprofloxacin may result in overgrowth of nonsusceptible organisms Repeated evaluation of the patient's condition and microbial susceptibility testing is essential If superinfection occurs during therapy, appropriate measures should be taken.

Information for Patients:

Patients should be advised that ciprofloxacin may be taken with or without meals The preferred time of dosing is two hours after a meal Patients should also be advised to drink fluids liberally and not take antacids containing magnesium or aluminum concomitantly or within two hours after dosing. Ciprofloxacin may cause dizziness or lightheadedness, therefore patients should know how they react to this drug before they operate an automobile or machinery or engage in activities requiring mental alertness or coordination.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Eight *in vitro* mutagenicity tests have been conducted with ciprofloxacin and the test results are listed below

- Salmonella/Microsome Test (Negative)
- *E coli* DNA Repair Assay (Negative)
- Mouse Lymphoma Cell Forward Mutation Assay (Positive)
- Chinese Hamster V_{79} Cell HGPRT Test (Negative)
- Syrian Hamster Embryo Cell Transformation Assay (Negative)
- *Saccharomyces cerevisiae* Point Mutation Assay (Negative)
- *Saccharomyces cerevisiae* Mitotic Crossover and Gene Conversion Assay (Negative)
- Rat Hepatocyte DNA Repair Assay (Positive)

Thus, two of the eight tests were positive, but the following three *in vivo* test systems gave negative results

- Rat Hepatocyte DNA Repair Assay
- Micronucleus Test (Mice)
- Dominant Lethal Test (Mice)

Long-term carcinogenicity studies in animals have not yet been completed

Pregnancy – Pregnancy Category C

Reproduction studies have been performed in rats and mice at doses up to six times the usual daily human dose and have revealed no evidence of impaired fertility or harm to the fetus due to ciprofloxacin In rabbits, as with most antimicrobial agents, ciprofloxacin (30 and 100 mg/kg orally) produced gastrointestinal disturbances resulting in maternal weight loss and an increased incidence of abortion No teratogenicity was observed at either dose After intravenous administration, at doses up to 20 mg/kg, no maternal toxicity was produced, and no embryotoxicity or teratogenicity was observed There are, however, no adequate and well-controlled studies in pregnant women SINCE CIPROFLOXACIN, LIKE OTHER DRUGS IN ITS CLASS, CAUSES ARTHR IMMATURE ANIMALS, IT SHOULD NOT BE USED IN PREGNANT WOMEN (SEE WARNINGS)

Nursing Mothers

It is not known whether ciprofloxacin is excreted in human milk, however, it is known that cipr excreted in the milk of lactating rats and that other drugs of this class are excreted in human milk Bec and because of the potential for serious adverse reactions from ciprofloxacin in nursing infants, a dec be made to discontinue nursing or to discontinue the drug, taking into account the importance of the mother

Pediatric Use

Ciprofloxacin should not be used in children because it causes arthropathy in immature ar WARNINGS)

ADVERSE REACTIONS

Ciprofloxacin is generally well tolerated During clinical investigation, 2,799 patients received 2,86 the drug Adverse events that were considered likely to be drug related occurred in 7.3% of cours related in 9.2%, and remotely related in 3.0% Ciprofloxacin was discontinued because of an adve 3.5% of courses, primarily involving the gastrointestinal system (1.5%), skin (0.6%), and central ner (0.4%)

The most frequently reported events, drug related or not, were nausea (5.2%), diarrhea (2.3 (2.0%), abdominal pain/discomfort (1.7%), headache (1.2%), restlessness (1.1%), and rash (1.1%)

Additional events that occurred in less than 1% of ciprofloxacin courses are listed below Thos quinolones are italicized

- GASTROINTESTINAL *(See above)*, painful oral mucosa, oral candidiasis, dysphagia, intestinal gastrointestinal bleeding
- CENTRAL NERVOUS SYSTEM *(See above), dizziness, lightheadedness, insomnia, nightmare. tions, manic reaction, irritability, tremor, ataxia, convulsive seizures, lethargy, drowsiness, malaise, anorexia, phobia, depersonalization, depression, paresthesia*
- SKIN/HYPERSENSITIVITY *(See above), pruritus, urticaria, photosensitivity, flushing, f angioedema, edema of the face, neck, lips, conjunctivae or hands,* cutaneous candidiasis, hyp tion, erythema nodosum
- SPECIAL SENSES *blurred vision, disturbed vision, (change in color perception, overbrightne decreased visual acuity, diplopia, eye pain, tinnitus, bad taste*
- MUSCULOSKELETAL *joint or back pain, joint stiffness,* achiness, neck or chest pain, flare-up
- RENAL/UROGENITAL *interstitial nephritis, renal failure,* polyuria, urinary retention, urethr vaginitis, acidosis
- CARDIOVASCULAR palpitations, atrial flutter, ventricular ectopy, syncope, hypertension, ang myocardial infarction, cardiopulmonary arrest, cerebral thrombosis
- RESPIRATORY epistaxis, laryngeal or pulmonary edema, hiccough, hemoptysis, dyspnea, br pulmonary embolism

Most of these events were described as only mild or moderate in severity, abated soon after t discontinued, and required no treatment

In several instances, nausea, vomiting, tremor, restlessness, agitation, or palpitations wer investigators to be related to elevated plasma levels of theophylline possibly as a result of a drug int ciprofloxacin

Adverse Laboratory Changes Changes in laboratory parameters listed as adverse events without re relationship

- Hepatic – Elevations of ALT (SGPT) (1.9%), AST (SGOT) (1.7%), alkaline phosphatase (0.8%) *serum bilirubin* (0.3%)
- Hematologic – eosinophilia (0.6%), leukopenia (0.4%), decreased blood platelets (0.1%), el platelets (0.1%), pancytopenia (0.1%)
- Renal – Elevations of Serum creatinine (1.1%), BUN (0.9%)
- CRYSTALLURIA, CYLINDRURIA, AND HEMATURIA HAVE BEEN REPORTED

Other changes occurring in less than 0.1% of courses were Elevation of serum gammaglutamyl elevation of serum amylase, reduction in blood glucose, elevated uric acid, decrease in hemoglo bleeding diathesis, increase in blood monocytes, and leukocytosis

OVERDOSAGE

Information on overdosage in humans is not available In the event of acute overdosage, the stoma emptied by inducing vomiting or by gastric lavage. The patient should be carefully observed and giv treatment Adequate hydration must be maintained. In the event of serious toxic reactions from hemodialysis or peritoneal dialysis may aid in the removal of ciprofloxacin from the body, partic function is compromised

DOSAGE AND ADMINISTRATION

The usual adult dosage for patients with urinary tract infections is 250 mg every 12 hours For complicated infections caused by organisms not highly susceptible, 500 mg may be administered e

Respiratory tract infections, skin and skin structure infections, and bone and joint infections m with 500 mg every 12 hours. For more severe or complicated infections, a dosage of 750 mg may be hours

The recommended dosage for infectious diarrhea is 500 mg every 12 hours

In patients with renal impairment, some modification of dosage is recommended (SEE D ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION).

HOW SUPPLIED

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CONVENIENT *B.I.D.* DOSAGE

Recommended dosage schedule

Infection Site*	Severity of Infection	Dos
Respiratory Tract* Bone and Joint* Skin/Skin Structure*	Mild/Moderate	500 mg
	Severe/Complicated	750 mg
Urinary Tract*	Mild/Moderate	250 mg
	Severe/Complicated	500 mg
Infectious Diarrhea*	Mild/Moderate/Severe	500 mg

* Due to susceptible strains of indicated pathogens. See indicated organisms in Brief Su

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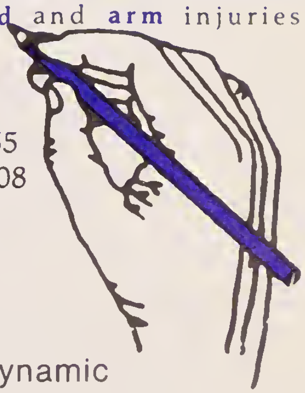

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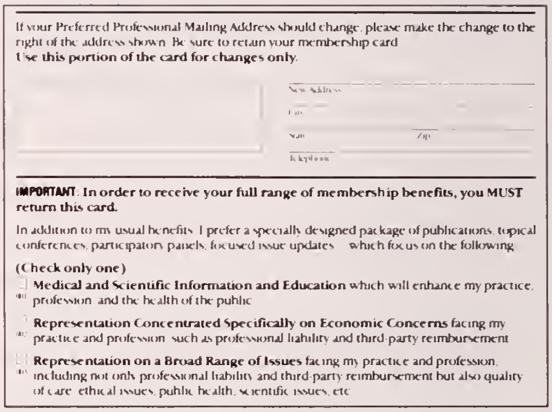

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Letters to the Editor

Medical Macro Economics-Reality vs Public Relations

To the Editor:

Politicians state that 10.5% of the GNP is going to medical care. Politicians are interested in political change. In reality, medical doctors control only a portion of these expenditures.

For example, in a recent year according to government statistics, $322 billion was expended for medical care. How much of this is really controlled by medical doctors? Only 60%.

- For analysis, medical costs not controlled by medical doctors should be subtracted.

Subtract

1. Nursing home care	$ 27 billion
2. Dental services	$ 20 billion
3. Chiropractors et. al.	$ 21 billion
4. Eyeglasses	$ 6 billion
5. Medical research	$ 8 billion
6. Medical construction	$ 8 billion
7. Government Public Health	$ 9 billion
8. "Malpractice" insurance	$ 3 billion
9. Government hospitals	$ 25 billion
10. Defensive medicine – Physicians (1)	$ 7 billion
11. Defensive medicine – Hospitals (2)	$7.5 billion

- These costs are more controlled by medical doctors.

Remaining Costs

1. Private hospitals	$103 billion
2. Physicians	$ 53 billion
3. Net costs health insurance	$ 13 billion
4. Drug preparations (3)	$ 12 billion
TOTAL	$181 billion

Debating health care costs means arguing over 5.6% of the GNP, *not* the heralded 10.5%.

Comparisons

- These amounts are spent annually by Americans for:

1. Education – elementary, secondary, college	$197 billion
2. Tobacco	$ 25 billion
3. Alcohol	$ 50 billion
4. Personal – legal services	$ 19 billion
5. Business – legal services (4)	$ 25 billion
6. Insurance	$ 41 billion

- Suggestions to decrease medical costs:

1. Reverse the 15 year process of federal government bureaucratization of American hospitals—costs 5% = $5 billion per year.
2. Control the civil court system which results in financial costs in the medical field alone estimated at $3+7+8 = $18 billion per year.
3. Continue public health push to decrease alcohol, tobacco, unnecessary vitamins ($3 billion per year).

You can help your employees by exploring substantial medicare supplemental insurance after the employee's retirement. Of total lifetime medical expenses, 80% occur in the last two years of life. The medicare system is staggering under this reality and is currently avoiding payment of charges for appropriate services by blaming doctors or hospitals and by erecting paperwork barriers to delay or prevent payment. This strategy will only work for a limited time.

Paul M. Worrell, M.D.
Anchorage, AK 99508

REFERENCES

1. JAMA, 1987;257:2776-2781.
2. Author's estimate.
3. Author's estimate; $10 billion is also spent on over the counter drugs.
4. Author's estimate.

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The following is a brief summary.

Contraindications: *There are no known contraindications to the use of 'Tagamet'.*

Precautions: *While a weak antiandrogenic effect has been demonstrated in animals, 'Tagamet' has been shown to have no effect on spermatogenesis, sperm count, motility, morphology or in vitro fertilizing capacity in humans.*

In a 24-month toxicity study in rats at dose levels approximately 9 to 56 times the recommended human dose, benign Leydig cell tumors were seen. These were common in both the treated and control groups, and the incidence became significantly higher only in the aged rats receiving 'Tagamet'.

Rare instances of cardiac arrhythmias and hypotension have been reported following the rapid administration of 'Tagamet' HCl (brand of cimetidine hydrochloride) injection by intravenous bolus.

Symptomatic response to 'Tagamet' therapy does not preclude the presence of a gastric malignancy. There have been rare reports of transient healing of gastric ulcers despite subsequently documented malignancy.

Reversible confusional states have been reported on occasion, predominantly in severely ill patients.

'Tagamet' has been reported to reduce the hepatic metabolism of warfarin-type anticoagulants, phenytoin, propranolol, chlordiazepoxide, diazepam, lidocaine, theophylline and metronidazole. Clinically significant effects have been reported with the warfarin anticoagulants; therefore, close monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when 'Tagamet' is administered concomitantly. Interaction with phenytoin, lidocaine and theophylline has also been reported to produce adverse clinical effects.

However, a crossover study in healthy subjects receiving either 'Tagamet' 300 mg. q.i.d. or 800 mg. h.s. concomitantly with a 300 mg. b.i.d. dosage of theophylline (Theo-Dur®, Key Pharmaceuticals, Inc.),

men, particularly in subjects aged 54 years and older. Data beyond ten days are not available. (Note: All patients receiving theophylline should be monitored appropriately, regardless of concomitant drug therapy.)

Lack of experience to date precludes recommending 'Tagamet' for use in pregnant patients, women of childbearing potential, nursing mothers or children under 16 unless anticipated benefits outweigh potential risks; generally, nursing should not be undertaken in patients taking the drug since cimetidine is secreted in human milk.

Adverse Reactions: *Diarrhea, dizziness, somnolence, headache, rash. Reversible arthralgia, myalgia and exacerbation of joint symptoms in patients with preexisting arthritis have been reported. Reversible confusional states (e.g., mental confusion, agitation, psychosis, depression, anxiety, hallucinations, disorientation), predominantly in severely ill patients, have been reported. Gynecomastia and reversible impotence in patients with pathological hypersecretory disorders receiving 'Tagamet', particularly in high doses, for at least 12 months, have been reported. Reversible alopecia has been reported very rarely. Decreased white blood cell counts in 'Tagamet'-treated patients (approximately 1 per 100,000 patients), including agranulocytosis (approximately 3 per million patients), have been reported, including a few reports of recurrence on rechallenge. Most of these reports were in patients who had serious concomitant illnesses and received drugs and/or treatment known to produce neutropenia. Thrombocytopenia (approximately 3 per million patients) and a few cases of aplastic anemia have also been reported. Increased serum transaminase and creatinine, as well as rare cases of fever, interstitial nephritis, urinary retention, pancreatitis and allergic reactions, including hypersensitivity vasculitis, have been reported. Reversible adverse hepatic effects, cholestatic or mixed cholestatic-hepatocellular in nature, have been reported rarely. Because of the predominance of cholestatic features, severe parenchymal injury is considered highly un-*

hepatic fibrosis in a patient receiving 'Tagamet' has been reported.

How Supplied: Tablets: *200 mg. tablets in bottles of 100; 300 mg. tablets in bottles of 100 and Single Unit Packages of 100 (intended for institutional use only); 400 mg. tablets in bottles of 60 and Single Unit Packages of 100 (intended for institutional use only), and 800 mg. Tiltab® tablets in bottles of 30 and Single Unit Packages of 100 (intended for institutional use only).*

Liquid: *300 mg./5 ml., in 8 fl. oz. (237 ml.) amber glass bottles and in single-dose units (300 mg./5 ml.) in packages of 10 (intended for institutional use only).*

Injection:

Vials: *300 mg./2 ml. in single-dose vials, in packages of 10 and 30, and in 8 ml. multiple-dose vials, in packages of 10 and 25.*

Prefilled Syringes: *300 mg./2 ml. in single-dose prefilled disposable syringes.*

Plastic Containers: *300 mg. in 50 ml. of 0.9% Sodium Chloride in single-dose plastic containers, in packages of 4 units. No preservative has been added.*

ADD-Vantage®* Vials: *300 mg./2 ml. in single-dose ADD-Vantage® Vials, in packages of 25.*

Exposure of the premixed product to excessive heat should be avoided. It is recommended the product be stored at controlled room temperature. Brief exposure up to 40°C does not adversely affect the premixed product.

'Tagamet' HCl (brand of cimetidine hydrochloride) injection premixed in single-dose plastic containers is manufactured for SK&F Lab Co. by Travenol Laboratories, Inc., Deerfield, IL 60015.

** ADD-Vantage® is a trademark of Abbott Laboratories.*

BRS-TG:L73B *Date of issuance Apr. 1987*

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PLANDEX 35201

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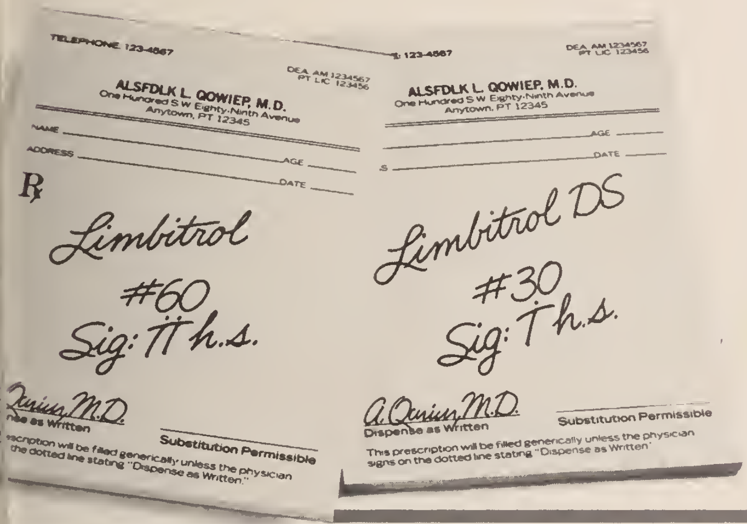
- 74% of patients experienced improved sleep after the first *h.s.* dose[1]
- First-week improvement in somatic symptoms[1]
- 50% greater improvement with Limbitrol in the first week than with amitriptyline alone[2]



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ALSFDLK L. QOWIEP, M.D.
One Hundred S W Eighty-Ninth Avenue
Anytown, PT 12345

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DATE

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Limbitrol®

Each tablet contains 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt) Ⓒ

Limbitrol® DS

Each tablet contains 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) Ⓒ

---

ferences: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Feighner VP, *al: Psychopharmacology 61:*217-225, Mar 22, 1979.

mbitrol® Ⓒ
anquilizer—Antidepressant
fore prescribing, please consult complete product information, a summary of which llows:

ntraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants; comitant use with MAOIs or within 14 days of monoamine oxidase inhibitors (then initiate utiously, gradually increasing dosage until optimal response is achieved); during acute recovery ase following myocardial infarction.

arnings: Use with caution in patients with history of urinary retention or angle-closure glauma. Severe constipation may occur when used with anticholinergics. Closely supervise cardioscular patients. Arrhythmias, sinus tachycardia, prolongation of conduction time, myocardial arction and stroke reported with tricyclic antidepressants, especially in high doses. Caution ients about possible combined effects with alcohol and other CNS depressants and against zardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving).

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations. Consider possibility of pregnancy when instituting therapy.**

hdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazees (see Drug Abuse and Dependence).

cautions: Use cautiously in patients with a history of seizures, in hyperthyroid patients, se on thyroid medication, patients with impaired renal or hepatic function. Because of suicidal ation in depressed patients, do not permit easy access to large quantities of drug. Periodic liver ction tests and blood counts recommended during prolonged treatment. Amitriptyline may k action of guanethidine or similar antihypertensives. When tricyclic antidepressants are d concomitantly with cimetidine (Tagamet), clinically significant effects have been reported olving delayed elimination and increasing steady-state concentrations of the tricyclic drugs. of Limbitrol with other psychotropic drugs has not been evaluated; sedative effects may be itive. Discontinue several days before surgery. Limit concomitant administration of ECT to ential treatment. See Warnings for precautions about pregnancy. Should not be taken during nursing period or by children under 12. In elderly and debilitated, limit to smallest effective age to preclude ataxia, oversedation, confusion or anticholinergic effects. Inform patients to sult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: Most frequent: drowsiness, dry mouth, constipation, blurred vision, dizziness, bloating. Less frequent: vivid dreams, impotence, tremor, confusion, nasal congestion. Rare: granulocytopenia, jaundice, hepatic dysfunction. Others: many symptoms associated with depression including anorexia, fatigue, weakness, restlessness, lethargy.
Adverse reactions not reported with Limbitrol but reported with one or both components or closely related drugs: *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke. *Psychiatric:* Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania, increased or decreased libido. *Neurologic:* Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns. *Anticholinergic:* Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue. *Endocrine:* Testicular swelling, gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Other:* Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. Withdrawal symptoms also reported with abrupt amitriptyline discontinuation. Therefore, after extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

Overdosage: Immediately hospitalize patient. Treat symptomatically and supportively. I.V. administration of 1 to 3 mg physostigmine salicylate may reverse symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

How Supplied: *Double strength (DS) Tablets,* white, film-coated, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt), and *Tablets,* blue, film coated, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

ROCHE
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JUN 28

In the depressed and anxious patient

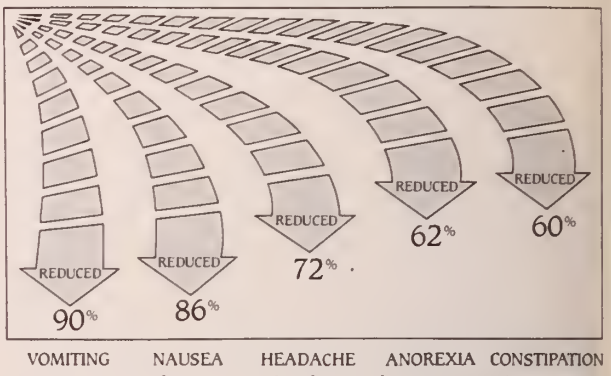
See Improvement In The First Week[1]...

And The Weeks That Follow

- 74% of patients experienced improved sleep after the first *h.s.* dose[1]
- First-week reduction in somatic symptoms[1]

Caution patients about the combined effects of Limbitrol with alcohol or other CNS depressants and about activities requiring complete mental alertness, such as operating machinery or driving a car. In general, limit dosage to the lowest effective amount in elderly patients.

Percentage of Reduction in Individual Somatic Symptoms During First Week of Limbitrol Therapy*



*Patients often presented with more than one somatic symptom.

Limbitrol®

Each tablet contains 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt) Ⓘⓥ

Limbitrol® DS

Each tablet contains 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) Ⓘⓥ

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ALASKA MEDICINE

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Volume 30, Number 4 July, August 1988

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(ciprofloxacin HCl/Miles)

A REVOLUTIONARY ORAL ANTIMICROBIAL WITH THE POWER OF PARENTERALS

- **Highly active *in vitro* against a broad range of gram-positive and gram-negative pathogens, including methicillin-resistant *Staphylococcus aureus* and *Pseudomonas aeruginosa****

- **For treatment of infections in the:**
  - **–lower respiratory tract†**
  - **–urinary tract†**
  - **–skin/skin structure†**
  - **–bones and joints†**

- **Convenient *B.I.D.* dosage – 250 mg, 500 mg and 750 mg tablets**

**In vitro* activity does not necessarily imply a correlation with *in vivo* results.

†Due to susceptible strains of indicated pathogens. See indicated organisms in Brief Summary.

CIPRO® SHOULD NOT BE USED IN CHILDREN OR PREGNANT WOMEN.

A history of hypersensitivity to ciprofloxacin is a contraindication to its use. A history of hypersensitivity to other quinolones may also contraindicate the use of ciprofloxacin.

Miles Inc.
Pharmaceutical Division
400 Morgan Lane
West Haven, CT 06516

Please see adjacent page of this advertisement for Brief Summary of Prescribing Information.

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C09327 MLR-261

Cipro®

TABLETS

(ciprofloxacin HCl/Miles)

■ 500 mg *B.I.D.* for most infections; 750 mg *B.I.D.* for severe or complicated infections.

BRIEF SUMMARY
CONSULT PACKAGE INSERT FOR FULL PRESCRIBING INFORMATION
INDICATIONS AND USAGE

Cipro® is indicated for the treatment of infections caused by susceptible strains of the designated microorganisms in the conditions listed below

Lower Respiratory Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Proteus mirabilis, Pseudomonas aeruginosa, Haemophilus influenzae, Haemophilus parainfluenzae*, and *Streptococcus pneumoniae*

Skin and Skin Structure Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Proteus mirabilis, Proteus vulgaris, Providencia stuartii, Morganella morganii, Citrobacter freundii, Pseudomonas aeruginosa, Staphylococcus aureus* (penicillinase and nonpenicillinase-producing strains), *Staphylococcus epidermidis*, and *Streptococcus pyogenes*

Bone and Joint Infections caused by *Enterobacter cloacae, Serratia marcescens*, and *Pseudomonas aeruginosa*

Urinary Tract Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Serratia marcescens, Proteus mirabilis, Providencia rettgeri, Morganella morganii, Citrobacter diversus, Citrobacter freundii, Pseudomonas aeruginosa, Staphylococcus epidermidis*, and *Streptococcus faecalis*

Infectious Diarrhea caused by *Escherichia coli* (enterotoxigenic strains), *Campylobacter jejuni, Shigella flexneri*,* and *Shigella sonnei** when antibacterial therapy is indicated

*Efficacy for this organism in this organ system was studied in fewer than 10 infections

Appropriate culture and susceptibility tests should be performed before treatment in order to isolate and identify organisms causing infection and to determine their susceptibility to ciprofloxacin Therapy with Cipro® may be initiated before results of these tests are known, once results become available appropriate therapy should be continued As with other drugs, some strains of *Pseudomonas aeruginosa* may develop resistance fairly rapidly during treatment with ciprofloxacin Culture and susceptibility testing performed periodically during therapy will provide information not only on the therapeutic effect of the antimicrobial agent but also on the possible emergence of bacterial resistance

CONTRAINDICATIONS

A history of hypersensitivity to ciprofloxacin is a contraindication to its use A history of hypersensitivity to other quinolones may also contraindicate the use of ciprofloxacin

WARNINGS

CIPROFLOXACIN SHOULD NOT BE USED IN CHILDREN OR PREGNANT WOMEN The oral administration of ciprofloxacin caused lameness in immature dogs Histopathological examination of the weight-bearing joints of these dogs revealed permanent lesions of the cartilage Related drugs such as nalidixic acid, cinoxacin, and norfloxacin also produced erosions of cartilage of weight-bearing joints and other signs of arthropathy in immature animals of various species (SEE ANIMAL PHARMACOLOGY SECTION IN FULL PRESCRIBING INFORMATION)

PRECAUTIONS

General.

As with other quinolones, ciprofloxacin may cause central nervous system (CNS) stimulation, which may lead to tremor, restlessness, lightheadedness, confusion, and very rarely to hallucinations or convulsive seizures Therefore, ciprofloxacin should be used with caution in patients with known or suspected CNS disorders, such as severe cerebral arteriosclerosis or epilepsy, or other factors which predispose to seizures (SEE ADVERSE REACTIONS)

Crystals of ciprofloxacin have been observed rarely in the urine of human subjects but more frequently in the urine of laboratory animals. Crystalluria related to ciprofloxacin has been reported only rarely in man, because human urine is usually acidic Patients receiving ciprofloxacin should be well hydrated, and alkalinity of the urine should be avoided. The recommended daily dose should not be exceeded Alteration of the dosage regimen is necessary for patients with impairment of renal function (SEE DOSAGE AND ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION)

Drug Interactions

Concurrent administration of ciprofloxacin with theophylline may lead to elevated plasma concentrations of theophylline and prolongation of its elimination half-life This may result in increased risk of theophylline-related adverse reactions If concomitant use cannot be avoided, plasma levels of theophylline should be monitored and dosage adjustments made as appropriate

Antacids containing magnesium hydroxide or aluminum hydroxide may interfere with the absorption of ciprofloxacin, resulting in serum and urine levels lower than desired, concurrent administration of these agents with ciprofloxacin should be avoided

Probenecid interferes with the renal tubular secretion of ciprofloxacin and produces an increase in the level of ciprofloxacin in the serum. This should be considered if patients are receiving both drugs concomitantly

As with other broad-spectrum antibiotics, prolonged use of ciprofloxacin may result in overgrowth of nonsusceptible organisms Repeated evaluation of the patient's condition and microbial susceptibility testing is essential If superinfection occurs during therapy, appropriate measures should be taken.

Information for Patients

Patients should be advised that ciprofloxacin may be taken with or without meals The preferred time of dosing is two hours after a meal Patients should also be advised to drink fluids liberally and not take antacids containing magnesium or aluminum concomitantly or within two hours after dosing Ciprofloxacin may cause dizziness or lightheadedness, therefore patients should know how they react to this drug before they operate an automobile or machinery or engage in activities requiring mental alertness or coordination.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Eight *in vitro* mutagenicity tests have been conducteu with ciprofloxacin and the test results are listed below

- Salmonella/Microsome Test (Negative)
- *E coli* DNA Repair Assay (Negative)
- Mouse Lymphoma Cell Forward Mutation Assay (Positive)
- Chinese Hamster V_{79} Cell HGPRT Test (Negative)
- Syrian Hamster Embryo Cell Transformation Assay (Negative)
- *Saccharomyces cerevisiae* Point Mutation Assay (Negative)
- *Saccharomyces cerevisiae* Mitotic Crossover and Gene Conversion Assay (Negative)
- Rat Hepatocyte DNA Repair Assay (Positive)

Thus, two of the eight tests were positive, but the following three *in vivo* test systems gave negative results

- Rat Hepatocyte DNA Repair Assay
- Micronucleus Test (Mice)
- Dominant Lethal Test (Mice)

Long-term carcinogenicity studies in animals have not yet been completed

Pregnancy – Pregnancy Category C

Reproduction studies have been performed in rats and mice at doses up to six times the usual daily human dose and have revealed no evidence of impaired fertility or harm to the fetus due to ciprofloxacin In rabbits, as with most antimicrobial agents, ciprofloxacin (30 and 100 mg/kg orally) produced gastrointestinal disturbances resulting in maternal weight loss and an increased incidence of abortion No teratogenicity was observed at either dose After intravenous administration, at doses up to 20 mg/kg, no maternal toxicity was produced, and no embryotoxicity or teratogenicity was observed There are, however, no adequate and well-controlled studies in pregnant women SINCE CIPROFLOXACIN, LIKE OTHER DRUGS IN ITS CLASS, CAUSES ARTH
IMMATURE ANIMALS, IT SHOULD NOT BE USED IN PREGNANT WOMEN (SEE WARNINGS)

Nursing Mothers

It is not known whether ciprofloxacin is excreted in human milk, however, it is known that ci
excreted in the milk of lactating rats and that other drugs of this class are excreted in human milk Be
and because of the potential for serious adverse reactions from ciprofloxacin in nursing infants, a de
be made to discontinue nursing or to discontinue the drug, taking into account the importance of t
mother

Pediatric Use

Ciprofloxacin should not be used in children because it causes arthropathy in immature
WARNINGS)

ADVERSE REACTIONS

Ciprofloxacin is generally well tolerated During clinical investigation, 2,799 patients received 2,8
the drug Adverse events that were considered likely to be drug related occurred in 7 3% of cou
related in 9 2%, and remotely related in 3 0% Ciprofloxacin was discontinued because of an ad
3 5% of courses, primarily involving the gastrointestinal system (1 5%), skin (0 6%), and central n
(0 4%)

The most frequently reported events, drug related or not, were nausea (5 2%), diarrhea (2
(2 0%), abdominal pain/discomfort (1 7%), headache (1 2%), restlessness (1 1%), and rash (1 1%)

Additional events that occurred in less than 1% of ciprofloxacin courses are listed below Th
quinolones are italicized

GASTROINTESTINAL *(See above)*, painful oral mucosa, oral candidiasis, dysphagia, intestin
gastrointestinal bleeding

CENTRAL NERVOUS SYSTEM *(See above), dizziness, lightheadedness, insomnia, nightma
tions, manic reaction, irritability, tremor, ataxia, convulsive seizures, lethargy, drowsine
malaise, anorexia, phobia, depersonalization, depression, paresthesia*

SKIN/HYPERSENSITIVITY *(See above), pruritus, urticaria, photosensitivity, flushing,
angioedema, edema of the face, neck, lips, conjunctivae or hands*, cutaneous candidiasis, h
tion, erythema nodosum

SPECIAL SENSES *blurred vision, disturbed vision, (change in color perception, overbright
decreased visual acuity, diplopia, eye pain, tinnitus, bad taste*

MUSCULOSKELETAL *joint or back pain, joint stiffness*, achiness, neck or chest pain, flare-
RENAL/UROGENITAL *interstitial nephritis, renal failure*, polyuria, urinary retention, ure
vaginitis, acidosis

CARDIOVASCULAR palpitations, atrial flutter, ventricular ectopy, syncope, hypertension, a
myocardial infarction, cardiopulmonary arrest, cerebral thrombosis

RESPIRATORY epistaxis, laryngeal or pulmonary edema, hiccough, hemoptysis, dyspnea,
pulmonary embolism

Most of these events were described as only mild or moderate in severity, abated soon afte
discontinued, and required no treatment

In several instances, nausea, vomiting, tremor, restlessness, agitation, or palpitations w
investigators to be related to elevated plasma levels of theophylline possibly as a result of a drug i
ciprofloxacin

Adverse Laboratory Changes Changes in laboratory parameters listed as adverse events without
relationship

Hepatic – Elevations of ALT (SGPT) (1 9%), AST (SGOT) (1 7%), alkaline phosphatase (0 8%
serum bilirubin (0 3%)

Hematologic – eosinophilia (0.6%), leukopenia (0 4%), decreased blood platelets (0 1%),
platelets (0 1%), pancytopenia (0.1%)

Renal – Elevations of Serum creatinine (1 1%), BUN (0 9%)

CRYSTALLURIA, CYLINDRURIA, AND HEMATURIA HAVE BEEN REPORTED

Other changes occurring in less than 0 1% of courses were Elevation of serum gammaglutam
elevation of serum amylase, reduction in blood glucose, elevated uric acid, decrease in hemo
bleeding diathesis, increase in blood monocytes, and leukocytosis

OVERDOSAGE

Information on overdosage in humans is not available. In the event of acute overdosage, the sto
emptied by inducing vomiting or by gastric lavage The patient should be carefully observed and g
treatment. Adequate hydration must be maintained In the event of serious toxic reactions fr
hemodialysis or peritoneal dialysis may aid in the removal of ciprofloxacin from the body, part
function is compromised

DOSAGE AND ADMINISTRATION

The usual adult dosage for patients with urinary tract infections is 250 mg every 12 hours F
complicated infections caused by organisms not highly susceptible, 500 mg may be administered

Respiratory tract infections, skin and skin structure infections, and bone and joint infections
with 500 mg every 12 hours For more severe or complicated infections, a dosage of 750 mg may b
hours

The recommended dosage for infectious diarrhea is 500 mg every 12 hours

In patients with renal impairment, some modification of dosage is recommended (SEE
ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION).

HOW SUPPLIED

Cipro® (ciprofloxacin HCl/Miles) is available as tablets of 250 mg, 500 mg, and 750 mg in bott
Unit-Dose packages of 100 (SEE FULL PRESCRIBING INFORMATION FOR COMPLETE INFORMAT

CONVENIENT *B.I.D.* DOSAGE

Recommended dosage schedule

Infection Site*	Severity of Infection	Dc
Respiratory Tract* Bone and Joint* Skin/Skin Structure*	Mild/Moderate	500 mg
	Severe/Complicated	750 mg
Urinary Tract*	Mild/Moderate	250 mg
	Severe/Complicated	500 mg
Infectious Diarrhea*	Mild/Moderate/Severe	500 mg

*** Due to susceptible strains of indicated pathogens. See indicated organisms in Brief**

**For further information, contact the Miles Information
1-800-642-4776.** In VA. call collect: 703-391-7888.

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ALASKA MEDICINE

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American Society for Circumpolar Health
Alaska Public Health Association

2401 E. 42nd Avenue, Suite 104, Anchorage, Alaska 99508

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Certifying Aeromedical Services in Alaska

by Mark S. Johnson[1]

ABSTRACT

The Alaska Department of Health and Social Services has developed three levels of aeromedical service certification—medical evacuation (medevac) service, critical care air ambulance service, and specialty aeromedical transport teams. To date, nine services serving Alaska have been certified at one or more levels. The department also contracted for development of aeromedical training courses, and several hundred pre-hospital and in-hospital medical personnel have received this training. The goal is to develop certified aeromedical services for pre-hospital medevacs in all regions to complement the excellent longer range air ambulance services already in existence.

BACKGROUND

Over the past decade, there has been explosive growth in the development of aeromedical services throughout the United States. Many hospitals, government agencies, and private companies have developed helicopter or fixed wing air ambulance services, most providing high quality aeromedical patient care. These services have greatly reduced transport times to hospital referral centers for patients from outlying areas or from smaller rural and suburban hospitals.

In Alaska, both Providence and Humana Hospitals have developed air ambulance services using primarily fixed wing aircraft due to the vast distances to rural hospitals and remote communities in this state. The North Slope Borough has purchased a helicopter for search and rescue or medevac missions, and a fixed wing air ambulance for faster, longer-range medical transportation. In Fairbanks, the 283rd Medical Detachment at Ft. Wainwright has been for many years designated a Military Assistance to Safety and Traffic (MAST) Unit, providing military helicopter ambulances for civilian medevac missions. In Glennallen, Cross Roads Medical Center (formerly Faith Hospital) has developed an aeromedical service to transport patients into Anchorage. And, in Seattle, the Airlift Northwest fixed wing air ambulance service routinely provides transportation for medical patients from Southeast Alaska.

All of these services have one thing in common—they use dedicated aircraft for aeromedical missions. However, many remote areas of Alaska are still several hours away from these dedicated aeromedical services. Consequently, most pre-hospital transportation needs of emergency medical patients in these remote areas cannot be served adequately by aeromedical services based hundreds of miles away.

To address these and other aeromedical issues in Alaska, in 1983 the Alaska Advisory Council on Emergency Medical Services and the Emergency Medical Services Section (EMS), Alaska Division of Public Health, Department of Health and Social Services organized a Medevac Task Force to study needs and to develop recommendations for aeromedical services in Alaska. After approximately 18 months of study and debate, this task force recommended that an air ambulance/medevac service certification process be established which does not necessarily require full-time, dedicated aircraft or full-time, dedicated flight crews. The task force also recommended that a statewide medevac escort training program be established.

Following these recommendations, in September 1985, the state EMS Section adopted certification regulations for Medevac Services, Critical Care Air Ambulance Services, and Specialty Aeromedical Transport Teams (1). The Purpose Section of these regulations stated, "It is not the intent of (these regulations) to prohibit transportation of emergency medical patients via any private aircraft, air taxi service, commercial airline, or government aircraft when it is in the best interests of the patient and no certified service is available within a reasonable period of time, as determined by the medical attendant with the patient. Rather, the intent is to prescribe minimum standards for air medevac services, air ambulance services, or specialty aeromedical transport teams that provide, offer, or advertise to provide emergency medical care that includes advanced life support services and transportation of sick or injured patients as a routine service, or to prescribe standards for basic life support services that choose to become certified."

CERTIFICATION REQUIREMENTS

The minimum basic requirements for Alaska's three different levels of certified aeromedical services are defined as follows:

1) **Medevac Service**
   a) Have a minimum of one certified EMT-I or higher level certified or licensed medical personnel to accompany each patient;

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b) Have appropriate medical equipment to provide emergency patient care within the skill levels of available medical personnel; and

c) Have a physician sponsor.

2) **Critical Care Air Ambulance Service**

a) Have a minimum of one licensed paramedic, registered nurse, or higher level licensed medical personnel to accompany each patient;

b) Have available, for all patients being transported, appropriate medical equipment meeting at least the minimum requirements recommended by the American College of Surgeons (2);

c) Have a physician medical sponsor who is either board certified or board eligible for, or has other department approved credentials showing a defined interest in, critical care or aerospace medicine;

d) Have written procedures for handling all categories of patients; and

e) Have available, 24-hours a day, seven-days a week, aircraft meeting the specifications listed in the U.S. Department of Transportation Air Ambulance Guidelines, including a heating system, air-to-ground and air-to-air communications, interior lighting for patient care, pressurization (if necessary for the patient), and adequate space for proper patient care (3).

3) **Specialty Aeromedical Transport Team**

a) Have a minimum of one licensed paramedic or registered nurse, or higher level licensed medical personnel, with specialized training for the category of patients the team is certified to transport (e.g. high risk infants);

b) Have available appropriate, department approved medical equipment for the category of patients to be transported;

c) Have a physician medical director who is board certified or board eligible in the medical specialty for which the team is certified; and

d) Have written procedures for handling all categories of patients to be escorted.

Note:

Each service may apply for more than one certificate. For example, the North Slope Borough has a medevac and a critical care air ambulance certificate and Providence Hospital's Life Guard Alaska has a critical care air ambulance certificate and a specialty aeromedical transport team certificate for its perinatal transport team.

In addition to the above requirements, all certified services must:

1) Use an aircraft operator in compliance with applicable Federal Aviation Administration regulations (4);

2) Have available aircraft with proper restraining devices for patients, litters, and equipment;

3) Be available 24-hours a day, seven-days a week, except when flying conditions are unsafe or when members of the service are busy with another medical emergency;

4) List in all advertisements the level of certified or licensed medical personnel available;

5) Test all equipment in the airborne environment to determine if it works as designed at high altitudes and doesn't interfere with aircraft operations;

6) Provide state approved medevac or aeromedical training, as appropriate, to all medical escorts and physician sponsors or medical directors; and

7) Complete a state approved in-flight patient care form for each patient transported.

Physician sponsors and medical directors also are required to approve protocols and medical standing orders, provide periodic review of patient care, and advise on the medical requirements of patient transportation in the airborne environment.

Aeromedical service teams which do not own or lease dedicated aircraft must show written agreements with aircraft operators that aircraft and pilots will be available when needed.

To ensure that aircraft are always available, due to the fact that air taxi operators charter aircraft for other missions besides EMS, certified medevac or air ambulance teams may have written agreements with two or more aircraft operators. This can be especially appropriate in areas where different types of aircraft may be needed for different types of missions.

To become re-certified, the medical attendants and physician sponsor/directors of medevac and air ambulance services must have at least 16 hours of department approved aeromedical training.

AEROMEDICAL TRAINING

The State EMS Section contracted with Southcentral Health Planning and Development, Inc., based in Anchorage, to develop aeromedical training manuals, including:

1) An Escort Training Manual;
2) Instructors Guide;
3) Planning and Procedures; and
4) Resources and Referring Physicians (5).

These manuals, along with a packet of instructor's slides, can be ordered from Southern Region Emergency Medical Services Council, Inc., also based in Anchorage.

FUTURE GOALS

So far, the Norton Sound Health Corporation in Nome, the Pond Reef Medi-Flite team in Ketchikan, and the Central Peninsula Medevacs team in Soldotna are the only certified aeromedical services in Alaska which don't have their own dedicated aircraft. Our goal is to get certified medevac teams organized in regional and subregional centers throughout the state, because we believe that certified teams can provide more consistent, quality pre-hospital aeromedical patient care and transportation than noncertified teams or medevac escorts which do not meet all of these standards.

A 1983 study of medevacs in Alaska, which was contracted for the EMS Section, showed that there were approximately 1,400 pre-hospital and 1,400 interhospital aeromedical transports per year throughout Alaska (6). Therefore, it seems that new medevac services can be organized and certified in places such as Kotzebue or Bethel without adversely affecting the economic viability of the medevac and critical care air ambulance services already in existence. For example, a certified medevac team from Bethel could transport patients from surrounding villages into the Yukon-Kuskokwim Regional Hospital and, if more specialized treatment is needed, one of the critical care air ambulance services from Anchorage could subsequently transport the same patients to hospital referral centers. Hopefully, some day soon all Alaskans will have timely access to appropriately trained and equipped aeromedical services no matter where they may live in the state.

For more information about aeromedical service certification in Alaska, contact the Emergency Medical Services Section, Division of Public Health, Department of Health and Social Services, P.O. Box H-06C, Juneau, Alaska 99811 (907-465-3027).

ALASKA AIR SERVICES CERTIFIED AS "OUTSIDE HOSPITALS" EMERGENCY MEDICAL SERVICES ACCORDING TO 7 AAC 23.310 — 7 AAC 26.400 AS OF 01/22/88:

MEDIVAC SERVICES (5)

Central Peninsula Emergency Medical Service*
M.A.S.T., 283rd Medical Detachment**
North Slope Borough Search and Rescue*
Norton Sound Health Corporation**
Temsco Helicopters, Inc./Pond Reef Medi-Flight*

CRITICAL CARE AIR AMBULANCE SERVICES (5)

Air Ambulance, Humana Hospital - Alaska*
Airlift Northwest*
Cross Roads Medical Center**
LifeGuard Alaska - Providence Emergency Transport*
North Slope Borough Search and Rescue*

SPECIALTY AEROMEDICAL TRANSPORT TEAMS (1)

LifeGuard Alaska - Providence Emergency Transport (Perinatal)*

*Expiration Date 12/31/89
**Expiration Date 12/31/88

REFERENCES

1. Alaska Administrative Code, Register 95, Health and Social Services, 7AAC 26.310 — AAC 26.400, "Medevac Services, Critical Care Air Ambulance Services, and Specialty Aeromedical Transport Teams," October, 1985.
2. American College of Surgeons, Hospital Resources Document, Appendix B, "Air Ambulance Operations," 1985.
3. U.S. Department of Transportation, National Highway Traffic Safety Administration, and the American Medical Association Commission on Emergency Medical Services, "Air Ambulance Guidelines," 1986.
4. 14 Code of Federal Regulations, Section 91 or part 135 of Federal Aviation Administration Regulations.
5. Johnson-Joseph L., Kelso L, and Marshall L. Aeromedical evacuations in Alaska: An Escort Training Manual, Instructors Guide, Planning and Procedures, and Resources for Referring physicians. The Alaska Medevac Escort Training Project, Southcentral Health Planning and Development, Inc., Anchorage, Alaska 1985.
6. Southcentral Health Planning and Development, Inc., "Alaskan Medevacs: Descriptive study, identification of problems, and possible solutions," Anchorage, Alaska, May, 1983.

Preliminary Trauma Study, 1987

by Steven J. Kilkenny, M.D., F.A.C.S.[1]

The leading causes of death, disability, and financial loss in Alaska are traumatic injuries. The leading cause of mortality is injury, documented by a recent report by the Alaska Division of Public Health (1). However, the incidence of nonlethal injury is poorly documented. Senate Bill 10 (2), introduced in the United States Senate by Senators Cranston, Gore, and Kennedy, will attempt to mandate the regulation of trauma activity in the near future. If passed, the bill will, in addition to other provisions, require development of a state emergency medical service and a regional trauma system including the designation of trauma centers, the development of quality assurance issues with regards to treatment of trauma, and the establishment of some form of trauma registry.

Further federal legislation in the form of H.R. 3133 (3), sponsored by the House Energy and Commerce Committees, will address trauma care and emergency medical services planning and development. Although S. 10 and H.R. 3133 differ on some details, they share four basic goals: Establishing a national clearinghouse on EMS information, improving emergency communication systems, establishing a grant program to fund the development and operation of regional trauma care networks, and conducting studies on the financial and societal impact of trauma.

In an effort to introduce the concept and encourage the formation of a trauma registry, 18 of the major emergency rooms in the state of Alaska were polled for the months of March, April, and May 1987 with regard to their trauma statistics. Key individuals in those emergency rooms were asked to review their data and report them. This information is provided in the following table. All transfers originated in the emergency department and all deaths occurred in the emergency room prior to admission.

Although this is a very brief and incomplete survey, it does give us some information with which to compare the utilization of trauma resources in the state. Currently, Southern Region Emergency Medical Services, under the direction of Tom Scott, is developing a trauma registry pilot project involving five hospitals in Southcentral Alaska. A trauma registry funding proposal was submitted to the Highway Safety Planning Agency, Department of Public Safety in May 1987 and was approved for funding for one year beginning in October 1987. A trauma registry will be an important step in the development of a more organized and comprehensive trauma system for the state of Alaska, because it will provide data on the types, locations, severity and treatment of traumatic injuries throughout the state. It will also help medical institutions address quality assurance items and risk management issues as they relate to trauma care.

Preliminary Trauma Survey 1987

Number of Trauma Admissions for a Three-month Period

Hospital	March	April	May	Deaths 3mo period occurring in E.R.	Transfers directly from E.R.
ANMC (Anchorage)	14	21	15		
Barrow PHS	12 total adm.				
Bartlett Memorial (Juneau)	17	14	28	1	1
Bristol Bay Area (Dillingham)	6	5	9		1
Central Peninsula (Soldotna)	35	30	35		20
Fairbanks Memorial	40	30	40	2	
Humana Alaska (Anchorage)	33	27	30	3	
Ketchikan General	10	11	11		
Kotzebue PHS	12 total adm.			4	1
Norton Sound Regional (Nome)	3	2	5	2	
Petersburg General	8	5	13		5
Providence (Anchorage)	81	77	97	3	
Seward General	7 total adm.				
Sitka Community	7	2	5		
South Peninsula (Homer)	4 total adm.				4
Valdez Community	2	1	4		
Valley (Palmer)	13	14	14		
Yukon-Kusk. Delta Reg. (Bethel)	15	4	12	1	9
TOTAL	**296**	**255**	**329**	**16**	**41**

REFERENCES

1. Mortality and Morbidity Study, Section of Epidemiology, Division of Public Health, Department of Health and Social Services, State of Alaska, Anchorage, 1985.
2. S. 10. A bill to amend the Public Health Service Act to improve emergency medical services and trauma care, and for other purposes; Senators Cranston, Kennedy, and Gore, 100th Congress, 1st Session.
3. H.R. 3133. Trauma Care and Emergency Medical Services Planning and Development Act, Rep. James Bates, 100th Congress, 1st Session.

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[1] Chairman, Alaska ACS Committee on Trauma.

Advanced Trauma Life Support in Alaska

by Steven J. Kilkenny, M.D., F.A.C.S.[1]

ABSTRACT

Two hundred former ATLS course participants were mailed a survey to evaluate retrospectively their use of skills and treatments taught in ATLS courses in Alaska. The survey also addressed their perception of trauma care in Alaska. One hundred and twenty-seven completed questionnaires were returned and evaluated. Differences in responses due to urban versus bush practice, specialty, practice type, frequency of multiple trauma management, and number of cases treated monthly have been analyzed. The purpose of this study was to evaluate the effect of the ATLS course on trauma care, to determine the frequency of use of the various procedures and skills taught in the ATLS course, and to develop some basic data with regard to trauma care in Alaska. Given the small sample size, apparent trends may not be representative of all trauma care providers.

INTRODUCTION

Trauma is the leading cause of all deaths in Alaska. A report published by the Alaska State Division of Public Health indicated that 70% of all deaths in Alaskans under 50 years of age were due to injuries (1). Alaska is a very young state—88.6% of the population is under the age of 50 years (2).

Recognizing that trauma is a surgical disease, often called the neglected disease of modern society, the American College of Surgeons Committee on Trauma has developed and sponsored the Advanced Trauma Life Support (ATLS) Course for physicians. The course, developed by the Lincoln Medical Education Foundation and the Southeast Nebraska Emergency Medical Services, was originally field tested in 1978 (3). The American College of Surgeons Committee on Trauma adopted the ATLS course in 1979. The course has been standardized and further revisions have been made. The ATLS course does not present new concepts in trauma care; however, it does teach well-established treatment and approaches to trauma care in a systematic manner, suggesting to the physician a concise method of establishing assessment and management priorities.

The ATLS course was introduced in Alaska in December 1981 when Dr. George Longenbaugh taught the first ATLS course in Sitka, Alaska. Dr. Longenbaugh was the first chairman of the State Committee on Trauma for the state of Alaska. The meritorious service award in ATLS was presented to him at the American College of Surgeons Clinical Congress meeting in October 1984 for his efforts in developing the ATLS course in Alaska (4). Since that time, a total of 19 ATLS courses have been held to educate approximately 312 physicians, 36% of the approximately 870 physicians licensed to practice in the state of Alaska (5). This is slightly misleading, however, because 17.3% of the respondents now live out of the state and several out-of-state physicians have come to Alaska to take the course (Table 1). In addition, 14 physician assistants have taken the formal course.

Funding for the ATLS course has come from the Alaska Division of Public Health, Emergency Services grants to regions, from the Alaska Area Native Health Service, and from course fees collected from participants.

This paper deals with a retrospective analysis of the ATLS course in Alaska in an attempt to determine the effect of the ATLS course on the treatment of trauma and to develop some basic data on the ATLS course itself.

METHODS

In January 1987, 200 former ATLS course participants were mailed written surveys about their involvement in and perceptions of trauma care in Alaska. From over 300 course participants, we were able to retrieve 200 valid and current addresses. We were unable to locate the remainder of the group. One hundred and twenty-seven questionnaires were completed and returned. Of the survey respondents, 55% had taken the ATLS course during 1983 to 1985, 19% took the course during 1981 to 1982, and 17% in 1986 to 1987. A few respondents had also taken a refresher course.

Participants were asked to define their practices, quantitate the number of times that they had used various skills taught in the ATLS course, and evaluate trauma care in their area. They also were asked to provide suggestions for limiting morbidity and mortality from trauma in Alaska.

RESULTS

In the following evaluation, differences in response due to urban versus bush practice, specialty, practice type, frequency of multiple trauma management, and

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number of cases treated monthly have been highlighted if they appear important.

Given the 64% response rate and the resultant very small sample size (in some cases less than 30), any apparent trends by urban versus rural practice, specialty, practice type, frequency of multiple trauma management, or number of cases treated monthly have been presented only to broaden a snapshot of the immediate data and should by no means be considered representative of all trauma care providers.

Location of Practice

Close to 83% of the ATLS course participants surveyed reside in Alaska, the remainder tend to be former Alaskan residents who have moved out of the state since taking the course. Of the general practice physicians, pediatricians, and physician assistants surveyed, 100% live in Alaska, as do 93% of the family practice physicians. General surgeons are the only group where non-Alaskans outnumber Alaskans (Table 1). This is due to the fact that six general surgeons from the lower 48 took an instructor course in Alaska so they could return to their respective areas as instructors. In fact all of the surgeons taking the ATLS course have done so as a prerequisite to becoming an instructor in the course.

Table 1. State of Residency by Specialty

Specialty	Alaska	Non-Alaska	Number
Family Practice	92.6%	7.4%	(54)
General Practice	100.0	—	(15)
General Surgery	40.0	60.0	(10)
Emerg. Medicine	66.7	33.3	(9)
Internal Medicine	66.7	33.3	(9)
Pediatrics	100.0	—	(6)
Phys. Assistants	100.0	—	(5)
Orthopaedics	66.7	33.3	(3)
Other	61.5	38.5	(13)
Total	82.7%	17.3%	(127)

Table 2. State of Residency by Bush versus Urban Physicians*

State	Total	Bush Physicians	Urban Physicians
Alaska	82.7%	91.4%	67.4%
Non-Alaska	17.3	8.6	32.6
(Number)	(127)	(81)	(46)

***Bush physicians are defined as having indicated a hospital bed capacity of 1-20 or 20-100, or by their addresses for those who did not report a hospital bed capacity. Urban physicians are defined as having indicated a hospital bed capacity of either 100-200 or 200+, or were also distinguished by their addresses in some cases.**

There are a total of 11 surgeon instructors in the state. Six are in private practice and five are employed by the Indian Health Service. The majority of general surgeons in Alaska have not been involved in the ATLS program, possibly because they feel adequately trained in the area of trauma. However, the American College of Surgeons considers the successful completion of the Advanced Trauma Life Support course a minimum indicator of commitment to trauma care by surgeons.

Of the 81 respondents practicing in rural areas, 91% still reside in Alaska compared to 67% of urban physicians (Table 2), reflecting the fact that the course has primarily been aimed at the bush physician.

Specialty

The largest professional group surveyed were family practitioner physicians (42%) and general practice physicians (12%). Close to 90% of the emergency medicine specialists are in private practice, as are 63% of the family practice physicians. Of the general practitioners, 73% are in government practice (Table 3). This is due to the fact that the majority of practice opportunities in the Alaskan bush are Public Health and Indian Health Service positions.

Urban and rural physicians appear to differ noticeably by specialty. Over 49% of the rural physicians are in family practice compared to 30% of urban physicians, and 15% of rural physicians are in general practice compared to 6.5% of urban physicians. Urban emergency medicine specialists (13%) and internists (15%) far outnumber their rural counterparts (emergency medicine specialists 4%, internists 3%, respectively). General surgery was somewhat more represented among urban (11%) than rural (6%) physicians. It must be noted that the vast majority of surgeons are located in urban centers (Table 4).

Table 3. Specialty by Practice Type

Specialty	Total	Private Practice	Government	Academic	Other	No Response	Number
Family Practice	42.5%	63.0%	35.2%	—	—	1.8%	(54)
General Practice	11.8	26.7	73.3	—	—	—	(15)
General Surgery	7.9	40.0	50.0	—	10.0%	—	(10)
Emergency Medicine	7.1	88.9	11.1	—	—	—	(9)
Internal Medicine	7.1	55.6	44.4	—	—	—	(9)
Pediatrics	4.7	50.0	50.0	—	—	—	(6)
Phys. Assistants	3.9	60.0	20.0	—	20.0	—	(5)
Orthopaedics	2.4	33.3	66.7	—	—	—	(3)
Other	10.2%	30.8%	46.1%	23.1%	—	—	(13)

Table 4. Specialty by Rural versus Urban Practice

Specialty	Total	Rural	Urban
Family Practice	42.5%	49.4%	30.4%
General Practice	11.8	14.8	6.5
General Surgery	7.9	6.2	10.9
Emerg. Medicine	7.1	3.7	13.0
Internal Medicine	7.1	2.5	15.2
Pediatrics	4.7	7.4	—
Physician Assistants	3.9	4.9	2.3
Orthopaedics	2.4	1.2	4.3
Other	10.2	6.2	17.4
No answer	—	3.7	—
(Number)	(127)	(81)	(46)

Board Certification

Over one-half of those surveyed are fully certified in a particular specialty. Certification is most prevalent among general surgeons (90%) and family practice physicians (63%) and is least prevalent among general practice physicians (13.3%) (Table 5).

Practice type

Fifty-three percent of the ATLS course participants are in a private practice while 42% work in government practice. Only 3% of those surveyed maintain academic appointments. More bush physicians (57%) are in private practice than in government practice (40%). Urban physicians are equally split between private (46%) and government practice (46%). Nine percent of the urban physicians have academic affiliations while no rural physicians have academic appointments. There is fairly even interest in the ATLS course by both private and government physicians both in the bush and urban setting (Table 6).

Hospital Bed Capacity

Those surveyed were most often associated with hospital sizes of 20 to 100 beds (38%) or 100 to 200 beds (24%). Fifty-seven percent of the respondents practice in rural hospitals (less than 100 beds) while 34% practice in urban facilities (100 beds or greater) (Table 7).

Table 6. Practice Type by Rural versus Urban Practice

Practice Type	Total	Bush Physicians	Urban Physicians
Private Practice	52.8%	56.8%	45.7%
Government	41.7	39.5	45.7
Academic	3.1	—	8.6
Other	1.6	2.5	—
No answer	0.8	1.2	—
(Number)	(127)	(81)	(41)

Table 7. Hospital Bed Capacity

Number of Beds	Total	Bush Hospitals	Urban Hospitals
Not hosp. based	6.3%	—	—
1-20	16.5	16.5%	—
20-100	37.8	37.8	—
100-200	24.4	—	24.4%
200+	7.9	—	7.9
no answer	7.1	5.5	1.6
(Number)	(127)	(81)	(46)

Frequency of Multiple Trauma Management

Seventeen percent of the physicians indicate that they "regularly" manage multiple trauma. About 53% of the respondents expressed that they "occasionally" manage multiple trauma. This is particularly true of the family practice physician (67%). Seventy-eight percent of the emergency medicine specialists "regularly" manage multiple trauma. Only 4% of family practitioners surveyed "regularly" manage multiple trauma. It appears that there are a wide range of "specialists" that regularly or occasionally manage trauma in Alaska (Table 8).

Urban physicians (22%) indicated that they "regularly" handle multiple trauma, while 12% of the bush physicians responded in a similar manner. Yet more bush physicians (57%) indicated that they "occasionally" manage multiple trauma than urban physicians (44%) (Table 9). Of those physicians who responded that they regularly manage trauma, 43% managed three to 10 cases per month, 27% managed 10 to 20 cases per month, 10% managed 20 to 40 cases a month. Of those "occasionally" managing trauma, 32% managed three to 10 cases a month, and 6% managed 10 to 20 cases a

Table 5. Board Certification by Specialty

Specialty	Fully Certified	Eligible for Certification	Not Certified	No Response	Number
Family Practice	63.0%	1.9%	11.1%	24.0%	(54)
General Practice	13.3	6.7	60.0	20.0	(15)
General Surgery	90.0	—	—	10.0	(10)
Emerg. Medicine	22.2	11.2	33.3	33.3	(9)
Internal Medicine	66.7	—	—	33.3	(9)
Pediatrics	50.0	—	16.7	33.3	(6)
Phys. Assistants	100.0	—	—	—	(5)
Orthopaedics	66.7	—	33.3	—	(3)
Other	38.5	—	38.5	23.0	(13)
Total	**54.3%**	**2.4%**	**19.7%**	**23.6%**	**(127)**

Table 8. Frequency of Multiple Trauma Management by Specialty

Specialty	Frequency Regularly	Occassionally	Rarely	Never	No Response	Number
Family Practice	3.7%	66.7%	22.2%	3.7%	3.7%	(54)
General Prac.	20.0	60.0	20.0	—	—	(15)
General Surgery	20.0	50.0	10.0	20.0	—	(10)
Emerg. Medicine	77.8	11.1	—	—	11.1	(9)
Internal Med.	—	44.4	44.4	11.2	—	(9)
Pediatrics	—	50.0	33.3	16.7	—	(6)
Phys. Assts.	20.0	60.0	20.0	—	—	(5)
Orthopaedics	66.7	—	33.3	—	—	(3)
Other	30.8	23.1	30.8	15.3	—	(13)
Total	**16.5%**	**52.8%**	**22.0%**	**6.3%**	**2.4%**	**(127)**

month. Those who "rarely" manage multiple trauma indicated that they treat less than three trauma cases per month (Tables 10 and 11). Emergency medicine specialists appear to treat more trauma cases monthly than other physicians, probably because they are primarily based in urban emergency rooms in larger hospitals that act as regional trauma centers in our state. Internists seem least active in trauma care.

Table 9. Frequency of Multiple Trauma Management

Frequency	Total	Bush Physicians	Urban Physicians
Regularly	16.5%	12.3%	21.7%
Occasionally	52.8	56.8	43.5
Rarely	22.0	22.2	23.9
Never	6.3	4.9	8.7
No answer	2.4	3.8	2.2
(Number)	(127)	(81)	(46)

Procedures Performed

The most frequent procedures performed by the responding course participants were: insertion of intravenous lines (90%), fracture immobilization (81%), intubation (80%), C-spine immobilization (76%), and transport of patients for definitive care (68%). The procedures least performed were peritoneal lavage (36%), pericardiocentesis (27%), insertion of central venous lines (24%), and cricothyroidotomy (9%).

Except for fracture immobilization, transport of patients for definitive care, and resuscitation of major burns, procedures taught in the ATLS course appear to be practiced slightly more by urban physicians than by bush physicians. This was particularly true of central venous placement (urban 33%, bush 19%), chest tubes insertion (urban 65%, rural 58%), and venous cut down (urban 52%, and rural 46%). On the other hand, a large percentage of rural physicians have performed transport of patients for definitive care (rural 74%, urban 59%),

Table 11. Monthly Trauma Caseload

Number of Cases	Total	Bush Physicians	Urban Physicians
0-3	59.1%	64.2%	45.7%
3-10	25.2	24.7	26.1
10-20	7.9	4.9	13.0
20-40	1.6	—	4.3
no answer	6.2	6.2	10.9
(Number)	(127)	(81)	(46)

Table 12. Procedures Performed since ATLS Course

Procedure	Total	Bush Physicians	Urban Physicians
Insertion of IV	89.8%	87.7%	93.5%
Fracture immobil.	81.1	84.0	76.1
Intubation	80.3	79.0	82.6
C-spine immobil.	76.4	77.8	78.3
Trans. pt. for defin. care	67.7	74.1	58.7
Chest tube	60.6	58.0	65.2
MAST pants	55.9	54.3	58.7
Treatment of major burn	55.1	58.1	50.0
Needle thoracentesis	50.4	51.9	47.8
Venous cutdown	48.0	45.7	52.2
Peritoneal lavage	36.2	34.6	39.1
Pericardiocentesis	26.8	24.7	30.4
CVP	23.6	18.5	32.6
Cricothyroidotomy	8.7	8.6	8.7
(Number)	(127)	(81)	(46)

Table 10. Managment of Multiple Trauma by Monthly Trauma Caseload

Frequency	Total	Number of Cases 0-3	3-10	10-20	20-40	no answer	Number
Regularly	16.5%	14.3%	42.9%	28.6%	9.5%	4.7%	(21)
Occasionally	52.8	56.7	32.8	6.0	—	4.5	(67)
Rarely	22.0	92.8	3.6	—	—	3.6	(28)
Never	6.3%	87.5%	—	—	—	12.5%	(8)

Table 13. Number of Procedures Performed since ATLS Course

		Number of Procedures					
Procedure	**None**	**1–10**	**11–20**	**21–50**	**51–100**	**100+**	**(Base)**
Insertion of IV	15.8%	13.2%	14.9%	18.4%	28.9%	8.8%	(114)
Fracture immobil.	24.3	34.0	10.7	13.6	15.5	1.9	(103)
Intubation	13.7	64.7	8.8	7.8	3.9	1.1	(102)
C–spine immobil.	24.7	48.5	4.1	8.2	13.4	1.1	(97)
Trans. of patients	15.1	38.4	12.8	17.4	15.1	1.2	(86)
Chest tube	18.2	72.7	5.2	3.9	—	—	(77)
MAST pants	9.9	81.7	2.8	1.4	4.2	—	(71)
Treat major burns	10.0	82.9	5.7	1.4	—	—	(70)
Needle thoracentesis	12.5	81.3	3.1	3.1	—	—	(64)
Venous cutdown	14.8	77.0	6.6	1.6	—	—	(61)
Peritoneal lavage	15.2	84.8	—	—	—	—	(46)
Pericardiocentesis	20.6	67.6	8.8	3.0	—	—	(34)
CVP	—	63.3	16.7	13.3	6.7	—	(30)
Cricothyroidotomy	—	100.0	—	—	—	—	(11)

fracture immobilization (bush 84%, urban 76%), and the initial treatment and resuscitation of major burns (bush 58%, urban 50%) (Table 12).

IV insertion by far is the procedure performed most often by the ATLS course participants surveyed. Since taking the ATLS course, 29% of the participants have performed between 51 to 100 IV insertions. Close to 10% of the respondents have performed over 100 IV insertions. We assumed that this skill was acquired prior to the ATLS course. However, it is definitely a skill that is practiced frequently and appropriately is reemphasized.

For each of the other procedures listed in the ATLS survey forms, only between one and 20 have commonly been performed by respective users since the ATLS course completion. Usage of cricothyroidotomy, peritoneal lavage, treatment of major burns, utilization of the military anti–shock trousers, and needle thoracentesis were more clustered in the one to 10 range than utilization of other procedures, suggesting even more limited use of these five procedures (Table 13).

Rating of Local Pre–Hospital Trauma Care

Almost 60% of those surveyed rated local pre-hospital trauma care as "good". Most noticeably, the general surgeons responding felt the strongest about this; 80% rated the pre–hospital trauma care as good.

A slightly higher percent of the urban physicians (63%) than the bush physicians (56%) rated local pre–hospital care as "good". More bush physicians (9%) than urban physicians (2%) felt that the quality of local pre–hospital care varies. Other rating levels were similar (Table 14).

The frequency of multiple trauma management and practice types generally did not influence perceptions of pre–hospital trauma care. Pre-hospital care for the most part is provided by volunteer emergency medical technicians in the bush areas. The urban areas are staffed by mostly salaried paramedics and EMTs. Training, education, and quality assurance in this area is governed by the regional and state emergency medical systems.

Rating of Local Surgical Consultants' Response to Trauma

Sixty–seven percent of those surveyed rate local surgical consultants response to trauma as "good," a perception which did not appear to be affected by practice type, specialty, or frequency of multiple trauma management.

More urban physicians (74%) rated local surgical consultants response to trauma as "good" compared to rural physicians (63%). Yet 24% of rural physicians rated local surgical consultants response to trauma as "adequate," as did 13% of the urban physicians. It must be noted that the surgical consultation available in most bush hospitals involves a long distance telephone call and cannot accurately be considered "local" surgical consultation (Table 15).

Table 14. Rating of Pre–Hospital Trauma Care by Rural versus Urban Practice

Rating	**Total**	**Bush**	**Urban**
Good	58.3%	55.6%	63.0%
Adequate	29.9	30.9	28.3
Poor	3.1	3.7	2.2
Varies	6.3	8.6	2.2
No resp.	2.4	1.2	4.3
(Number)	(127)	(81)	(46)

Table 15. Rating of Local Surgical Consultants' Response to Trauma

Rating	**Total**	**Bush**	**Urban**
Good	66.9%	63.0%	73.9%
Adequate	19.7	23.5	13.0
Poor	2.4	3.7	—
Varies	3.1	2.4	4.3
No avail.	2.4	3.7	—
No resp.	5.5	3.7	8.8
(Number)	(127)	(81)	(46)

Involvement in Trauma Accident Prevention

Over 60% of the respondents were involved in trauma/accident prevention. Although the number of physicians is quite small, prevention activities are most common among internists (78%) and least common among general surgeons (30%) (Table 16).

Table 16. Involvement in Trauma/Accident Prevention by Specialty

Specialty	Yes	No	Number
Family Practice	63.0%	37.0%	(54)
General Practice	53.3	46.7	(15)
General Surgery	30.0	70.0	(10)
Emergency Medicine	66.7	33.3	(9)
Internal Medicine	77.8	22.2	(9)
Pediatrics	66.7	33.3	(6)
Physician Assistants	100.0	—	(5)
Orthopaedics	66.7	33.3	(3)
Other	53.8	46.2	(13)
Total	**62.2%**	**37.8%**	**(127)**

Urban and rural physicians appear equally involved in trauma/accident prevention. Moreover, practice types also did not seem to influence involvement in prevention activities. The actual type of involvement in trauma/accident prevention was not solicited.

Those who "rarely" or "never manage" multiple trauma are equally as active in trauma/accident prevention as those who are more frequently involved in multiple trauma management.

Willingness to Participate in Statewide Trauma Registry

Close to three quarters of the ATLS course participants indicated willingness to participate in a statewide trauma registry. This was particularly true of those who "regularly manage multiple trauma" (91%); and it was also characteristic of those who "occasionally" (70%) or "rarely" (71%) manage multiple trauma. Both rural and urban physicians were similarly willing to participate in a trauma registry.

Interest in a One-Day Refresher Course

Eighty percent of the respondents expressed interest in a one-day refresher course, particularly in the general practitioners (100%) and the physician assistants (100%). It should be noted that only 15 general practice physicians and five physician assistants were surveyed. General surgery physicians were least interested in a refresher course (40%) (Table 17). To date, there has been one refresher course held in Alaska with others to be scheduled as the need arises.

Table 17. Interest in One-Day Refresher Course by Specialty

Specialty	Yes	No	Number
Family Practice	83.3%	16.7%	(54)
General Practice	100.0	—	(15)
General Surgery	40.0	60.0	(10)
Emergency Medicine	77.8	22.2	(9)
Internal Medicine	77.8	22.2	(9)
Pediatrics	83.3	16.7	(6)
Physician Assistants	100.0	—	(5)
Orthopaedics	66.7	33.3	(3)
Other	69.2	30.8	(13)
Total	**80.3%**	**19.7%**	**(127)**

Interest in a one-day fresher course was higher among bush physicians (84%) than among urban physicians (74%) (Table 18).

Table 18. Interest in One-Day Refresher Course by Rural versus Urban Physicians

Response	Total	Bush Physicians	Urban Physicians
Yes	80.3%	84.1%	73.2%
No/no Response	19.7	15.9	26.8
(Number)	(127)	(81)	(46)

Suggestions for Limiting Traumatic Morbidity and Mortality in Alaska

Suggestions for limiting traumatic morbidity and mortality largely centered around reducing alcohol abuse, harsher and stricter enforcement of DWI laws, increased use of seatbelts, and reducing use of off-road vehicles by minors. It was generally felt that DWI laws should be extended to off-road vehicles (three-wheelers and ATVs), as well as boats. Preventive education programs relative to alcohol and mix of alcohol and vehicular operation, along with gun safety, fishing safety, and boating safety were also recommended (Table 19).

Table 19. Suggestions for Limiting Morbidity and Mortality (Selected Statistics)

Suggestion	Total	Rural	Urban
Eliminate alcohol in "bush"; reduce alcohol abuse in "bush"; more enforced DWI laws for off-road vehicles and boats	29.1%	32.1%	24.0%
More preventive education programs regarding alcohol, alcohol and vehicle operation, gun safety, hunting and fishing safety, boating safety	13.4	11.1	8.7
Age requirement; drivers license requirement for off-road vehicles; no kids on off-road vehicles	11.0	13.6	10.9
Mandatory seat belt usage	10.2	9.9	13.0
(Number)	(127)	(81)	(46)

DISCUSSION

It is obvious that the ATLS Course is of extreme importance in Alaska. Alaska is truly a land of superlatives—the largest, coldest, longest, highest, most isolated, and farthest north and west of all the states. It is one of the least populated states, has the highest point on the continent, and has more coastline than all of the other states combined.

However, these characteristics that make Alaska most impressive and unique to the visitor also make the delivery of health care one of the most challenging and most difficult tasks in the country. Geography and climate impose severe limitations on transportation and communication in Alaska. No road systems exist to link most of the more than 200 Alaskan communities in which live almost one-fourth of the state's population.

To receive basic medical care, it is frequently necessary for patients to fly as far as 500 miles to see a physician or dentist. Almost half of the population in the state of Alaska, or approximately 235,000 people, live in Anchorage, which is the major trauma referral center for the entire state. There are no cardiac or neurosurgical capabilities in any other city in Alaska. Of the 30 general surgeons in the state, 75% are located in the southcentral area around Anchorage. The majority of small bush hospitals with bed capacities ranging from six to 40 have no general surgical capabilities and no ability to treat major trauma. Most of the medical practitioners in these areas have had no formal education in trauma management. This necessitates the transfer of most of the major trauma either to the Anchorage area or to Fairbanks, which is the second largest city. Juneau, Sitka, and Ketchikan, the largest cities in southeast Alaska, all have general surgical capabilities. Occasionally, multiple trauma victims from southeast Alaska are transferred to Harborview Hospital in Seattle.

Stabilization and transport of major trauma injuries is very important in the state of Alaska. It almost seems as if the ATLS course was developed specifically for the needs of a state like Alaska. There is no doubt, from the comments made by the participants who responded that the ATLS experience in Alaska has had a meaningful role in influencing their attitudes on the management of trauma.

Currently there are efforts to establish a trauma care system in Alaska. The majority of ATLS physicians support the idea of a trauma registry and appear to be interested in prevention of trauma through legislation and education. The study clearly points out that the skills taught in the ATLS course are utilized. The ATLS course is I believe saving lives and substantially diminishing the morbidity from trauma. It is the goal of the Alaska Committee on Trauma to have all trauma care providers participate in the ATLS course on one level or another, as a minimum indication for their dedication to providing quality trauma care in the state of Alaska.

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Exercise Adherence: The Problems of Sticking with It

by Jay E. Caldwell, M.D., M.P.H., F.A.C.S.M.[1]

Little doubt lingers that exercise, or at least physical activity, is healthful for almost everyone. Getting our patients to be more active is another matter.

Though surveys often note that doctors do too little health and fitness urging, improving our nation's fitness will not come about simply by our bringing up the subject during a routine office visit. We will find ourselves preaching to the converted. The ones who would benefit most do not find their way into the medical community.

In the recently published text *Exercise Adherence*, Dorothy N. Knapp, Ph.D., Department of Medical Psychology, Oregon Health Sciences University, Portland, observes that much of our failure can be ascribed to our reliance on the "Health Belief Model" of behavioral change.

Because exercise is not a normative behavior, such as tooth brushing, and because there are few direct social or material benefits which accrue to those who do exercise, the medical profession has chosen to rely on the scare tactics of the Health Belief Model.

This model assumes first that we have a concern about our health, and that we are willing to accept some guidance for achieving improved health or minimizing poor health. The next assumption is that we recognize that there exists a threat of illness which may potentially inflict some form of bodily harm that will leave unwanted physical, social, or psychological footprints. Then, we must believe that the recommended intervention will work and that the side effects will not outweigh the benefits. Finally, we must believe that we are actually capable of performing the necessary regimen.

Realistically, the link between physical activity and illness avoidance is very loose. A sedentary person can point to many a fat slob who apparently is in perfectly good health. For that matter, most sedentary people are seemingly healthy. Faced with this harassment, it is just as reasonable for the couch potato to leave the arena completely in order to avoid further nagging.

Another problem with this Health Belief Model is that it creates for the sedentary person another "should." An imbalance of "shoulds" over "wants" underlies a desire for indulgence. The advertising line that runs, "you deserve a break today," provides the underlying message that even though a Big Mac is not particularly healthy, it is too much of a drudge to eat right all the time. So relax, "chill out," indulge.

This leads to cravings in which immediate gratification is sought. Once the "should" is broken, feelings of self-worth plummet, and our sense of mastery over our bodies is diminished. First guilt, then rationalization and denial flower; the pathway to relapse is greased.

It would be a better approach to identify for our patients some of the putative benefits from exercise that will result in more obvious and immediate gratification. In this way "wants" will be created, and from this positive base it will be much easier to achieve compliance.

The nonmedical benefits of exercise are vast. There is the opportunity for social interaction; in some cases this can have direct professional ramifications. It is not untrue that many important business contacts are made on the golf course or in the showers after a racquetball game.

Regular exercise adds to our ability to participate in family activities as disparate as softball and hiking. Exercise can thereby immeasurably improve family dynamics. Many will believe their appearance to improve, even before it has. There will be a sense of added physical prowess. Urges for competition can be carefully cultivated. Stress reduction is a given as long as the "should" effect does not occur.

Physical activity can substitute for a variety of other "shoulds." A good example would be the release from the shackles of a restrictive diet. The need for medication may be reduced or eliminated, this being particularly true in asthma, diabetes, and many forms of heart disease.

In managing dysfunctions of the musculoskeletal system, rehabilitation plays a crucial role. Nevertheless, it is sheer drudgery. There is probably little more tedious in all of existence than having to perform a daily back program, be they Williams flexions, McKenzie extensions, or today's vogue, the "neutral" back regimen. Such a prescription is one of the most odious "shoulds" going. Flare-ups are inevitable and the self-reproach that follows may be so detrimental as to drive reasonable patients into the hands of charlatans.

Were the patient encouraged to tackle a sport that would not obviously be contraindicated considering the medical or orthopedic limitations, a sport that could even fulfill a competitive drive or the fantasy of enhanced sexual prowess, a "want" will be created.

We all end up rebelling against our "shoulds," whereas we defend our "wants," often beyond reason. Nevertheless, for some, exercise is initially an unpleasant, inconvenient, and often uncomfortable new behavior. It is equally clear that almost everyone can find a level of physical activity that will end up not being unpleasant, and perhaps even satisfying.

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By piloting our patients first through the difficult waters of the habit acquisition phase while helping them develop the skills needed to avoid the shoals of the maintenance period, we will have discharged our duty as physicians admirably.

Exercise habits are influenced by physiological, psychological, and environmental factors. Yet it is only over the last that we can exert meaningful control. A behavior becomes established when there are sufficient cues which elicit it, and adequate reinforcement to entrain it. Although cues and reinforcement will initially come from the environment, ultimately they must become internalized.

There are thus three points in this process at which we can make changes: the cues, the behavior itself, and the consequences of that behavior. As physicians we tend to concentrate on the second. It is relatively simple to design exercise prescriptions for our patients or to advise them about shoes or community resources, but no matter how well-intentioned they are, without adequate behavioral training, the failure rate will be high.

It is crucial for the beginner to have adequate cues to start exercising. One of the simplest, and therefore best cues is regularity. If you exercise at the same time each day, this will be a natural cue.

Fixed schedules can backfire on you. If you have selected the lunch hour as your exercise period, you had better be sure that time is really yours. If lunch occurs at the whim of others, if the time or duration of lunch is variable depending on the work schedule, you had better beware. No matter how well-intentioned "brown bag conferences" are, most are not conducive to an exercise program.

Pairing exercise to another cue can be very helpful. A good example would be riding your stationary bicycle while watching a particular television program. It is important that the paired cue be a low frequency event so that it will stand out as special. If you plan to watch music videos or the news while riding your bike, there are too many opportunities during the day. The more alternatives you allow yourself, the less satisfactory will be that cue.

Logging records, even if after the fact, is an excellent cue. Establishing and periodically reviewing your weekly plan is a strong motivator. In such a journal you must also be sure to indicate why you didn't meet a goal. Later, that information may help you create more successful cues.

Preparation is another valuable cue. Having your exercise clothes or equipment laid out where it would be difficult not to trip over them is an excellent reminder. The more times you are reminded of your commitment, the easier it will be to remember.

If there are distracting cues, they need to be minimized. If you exercise late in the day and you find home a very relaxing place, it would be best not to stop by there on your way to the gym. The simple act of putting your clothes in your car in the morning is a good preparatory cue and minimizes distraction.

Reinforcement is the other aspect of behavior that needs to be addressed. The longer the chain between the action and the outcome, the less effective will be the feedback. Reinforcement can be either positive or negative. Behaviorists rarely agree, but on punishment they are united. It is an ineffective way to establish a behavior. Reinforcement needs to be positive, to be pleasing.

Some would argue that for many groups—especially the unfit, the obese, and the elderly—exercise has built-in punishments: embarrassment, imagined ridicule, and discomfort. Since the natural reaction to punishment is avoidance or escape, these patients may have a harder row to hoe.

One technique which has been used to counter such negative feedback is dissociation. Dissociation is the use of distractive coping techniques to tune in other more neutral cues and reinforcers. If embarrassment is central, then it is important to focus on the good you know must be taking place inside you. If it is your own physical discomfort that is disconcerting, then you need to tune into the sky and trees.

Reinforcement is much more than just a reward system. For example, self-monitoring is one of the most effective reinforcements. It requires the maintenance of a diary and the establishment of goals. Seeing miles accumulate and performances improve, goals being met, is one of the best ways to internalize reinforcement.

Reinforcement can take many forms. Simple praise or encouragement is very effective. The opportunity for new personal interactions can be quite satisfying. Although social reinforcement is valuable in the habit acquisition phase, it is self-limiting. After a while the new will not be so new, and comments will dwindle. Whatever changes we do make must be for ourselves, not for others.

Family and social support is another type of reinforcement. If your spouse approves of your exercise efforts and is willing to time-share the more mundane household responsibilities, such as cooking, cleaning, and childcare, it will make the time you spend away from your family more rewarding. If you have to fight the system all the time, failure is imminent.

Habit acquisition takes at least months. Thereafter comes the maintenance phase. It is during this period that lapses, and all the emotional fallout from them, must be managed. Lapses are inevitable. Improperly handled, they may drop the self-esteem of a recent convert to an all-time low.

Lapses come about because of an inadequate coping or avoidance response to a high risk situation. Because there will always be high risk situations, planning for them is necessary.

Identifying them is the first step, and the daily journal is the foundation. Role playing can be useful in the sense of "what will you do when...?"

For many, a group of friends, a basketball team, or a unique facility forms the heart of the cue and reinforcement system. What will you do when they're sick, the league season ends, or there's a power outage? Alaskan weather is another good example: what do you do when the roads are icy and it's dark at 4:30? You must have some notion of how to manage these and other high-risk situations.

In addition to planning for lapses by having alternate plans, other coping skills such as assertiveness training and time management may be valuable. Because slips are inevitable, a certain amount of cognitive restructuring will be helpful. Lapses need to be viewed as learning experiences, not as failures. In such a way a lapse may conceivably even raise self-esteem.

Failure can be mitigated also by structuring goals such that short term (days and weeks) ones are merely steps towards the important long-term goal. Goals need to be behavioral ones, not fixed outcomes. This is especially important in weight loss programs in which the real goal must be viewed as a permanent lifestyle change. Weight loss is then just a by-product.

Cultivating a belief that health will suffer if exercise does not occur is counterproductive. This Health Belief Model has been the strategy that public health officials as well as private physicians have long used to cajole the public and individual patients into a more vigorous lifestyle.

If, as physicians, we can provide for our patients concrete ideas about how to establish an active lifestyle so that it becomes a "want" rather than another burdensome "should," we will have taken a major step in improving their health. There is no better way to learn the nuts and bolts of providing such advice than to create such a program for ourselves.

It is our responsibility to be promoters of health, but often the road to that end will travel across rather unusual terrain. We must not fear exploring it.

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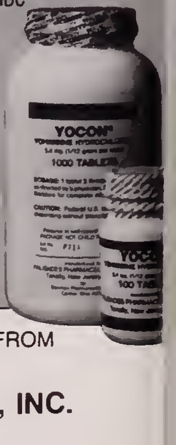

Electronic Information for Physicians: A New Dimension in Solving Traditional Problems

Part 1

by William A. Yasnoff, M.D., Ph.D.
American Medical Association

Physicians have a continual need for information. This is a natural consequence of the overwhelming and increasing quantity of biomedical knowledge required for the practice of medicine today. Traditional sources of this information include colleagues, books, and journals, both in the hospital and office environment. Thanks in part to the decreasing cost of computer equipment, a new, low-cost, more up-to-date source of information is now readily available to physicians: electronic information. This information may often be more convenient, more current, and more relevant than traditional sources. This article provides some examples of the types of electronic information which can be useful in addressing physician information needs. It focuses on AMA/NET, the AMA sponsored nationwide medical information network which is now available to physicians throughout the United States.

I. The Diagnostic Problem

Probably the most common problem that physicians face is making a diagnosis. In the overwhelming majority of cases, the signs and symptoms of a patient's presenting illness are clear and easily recognizable and the diagnosis is rapidly made. However there is a small percentage of cases which present puzzling combinations of findings. It is in these cases that physicians traditionally seek the help of colleagues, books and the recent literature. Colleagues, of course, are an excellent source of information. However, they are not always readily available. Books typically are limited in that their information is at least one year old, and is usually organized by disease, not signs and symptoms. Because of this, a diagnostic hypothesis must be formed before the information is accessible. Even books which focus on signs, symptoms, and differential diagnosis may not provide ready access to information relevant to a specific patient with an unusual combination of findings.

While the recent literature is more current than books, it is also organized by disease and is more difficult to access. Only a limited number of journals can be available in the physician's office. These will typically be organized chronologically, and finding information on a particular topic may be quite time consuming. A well organized and up-to-date reprint system may compensate for these problems somewhat.

The nearest medical library will undoubtedly have a much more comprehensive collection of journals and also more capabilities for locating relevant articles, for example using Index Medicus. However, the library may also not be very convenient for the physician.

How can electronic information help? AMA/NET provides two different types of information which can be of assistance in a diagnostic problem: 1) DxPlain, a diagnostic assistance program, and 2) various methods for searching and accessing the medical literature. The DxPlain system is unique to AMA/NET; it provides patient-specific diagnostic information. Upon entering DxPlain, information is requested regarding the age and sex of the patient, duration of the problem, and the signs, symptoms, and lab findings associated with this condition. This information is entered in a very natural form with opportunities for correcting spelling and other typographical errors. After entering all the information for the patient, DxPlain produces a list of diagnostic possibilities. This list is divided into common and rare diseases, and each disease shown is given a ranking according to the likelihood that it could be responsible for the observed patient signs and symptoms. DxPlain then will provide explanations describing those signs and symptoms that led to the inclusion of each diagnosis, other findings which are not explained, and additional findings which were not noted but would be expected in that particular condition. Thus, DxPlain can help the physician to both generate and "think through" the differential diagnosis in a puzzling case. It functions as a specialized, patient-specific differential diagnosis reference allowing rapid access to information which can be helpful in the diagnostic process.

Another important area where electronic information can be extremely useful is in search and retrieval of recent literature. AMA/NET provides several methods of accessing the medical literature. The first of these, is EMPIRES (Excerpta Medica Physicians Information Retrieval and Education Service), which indexes articles from 328 key clinical journals. Searching can be done in one of two ways: easy search and advanced search. In easy search, two keywords are entered and a search is performed for all references which pertain to both those keywords. An option is provided for restricting the search to the last six months. Very little (if any) knowledge with respect to searching techniques in databases is required to use easy search. In advanced search, as many keywords as desired may be entered, and combined in whatever way desired. Repeated searches can be done to narrow down the selections as needed. While advanced search is not difficult to use, it does require some knowledge of both searching techniques and the commands available. AMA/NET provides access to MEDLINE, the National Library of Medicine database of the world's biomedical literature, in two separate ways. First, access is provided by PaperChase, a friendly user interface developed at the Beth Israel Hospital in Boston. This program allows the physician with very little searching experience or knowledge of computers to construct a successful search of some complexity. Many physicians have found that it provides a fast and easy way to access the medical literature.

The National Library of Medicine's own user interface for MEDLINE, Grateful Med, is also available on AMA/NET. It provides a slightly different environment for helping the physician to develop and execute an appropriate search.

All three of these methodologies provide a useful way to access the most recent medical literature. Although the full text of articles found is not on-line for immediate reading, abstracts are available for the majority of the articles. Also, it is possible to order paper copies of any articles retrieved (up to five EMPIRES articles per month are available at no charge).

II. The Therapeutic Problem

The physician's approach to a therapeutic problem is very similar to the diagnostic, although there are some sources of information which are more directly helpful. As with diagnostic problems, the traditional options available are colleagues, books, and literature. If information is desired on a specific drug or a class of drugs, this can be obtained from a number of references which are often readily available. However, the often-asked question regarding the determination of the currently accepted therapy for a given disease is sometimes more difficult. Here again, electronic access to the most recent literature can be very helpful.

One of the key words allowed in literature searching is "review." This results in the ability to access a recent review article on any given condition. This can often result in rapid identification of currently acceptable therapy.

Another common therapeutic problem is assessing the possibility of drug interactions, especially for a patient on many different medications. The MEDICOM drug interaction database recently became available on AMA/NET. It allows entry and storage of specific patient drug profiles so that patients with difficult drug interaction problems can be easily reevaluated when therapy is changed. In addition to drug interactions, drug-food, drug-nutrient, and drug-disease, and potential allergic reactions are also reported. Each reaction has an associated level of severity. If desired, output can be limited to the most clinically significant reactions. This avoids one of the classic problems in consulting printed drug interaction references: namely, the hundreds of interactions listed which have no practical clinical significance.

Next time: Current Awareness, Electronic Communication with Colleagues, and more.

Providence Hospital film wins international award.

The John Muir Medical Film Festival has awarded Providence Hospital, Anchorage a First Place Gold Award in the "Public Relations - Long" category for their 50th anniversary slide show entitled "The Commitment Continues".

The historic slide presentation competed against 476 entries from 21 countries and was screened by leaders in medicine and media production from around the world at the Festival Awards ceremony June 25, 1988 in San Fransisco.

The 14 minute black and white slide presentation chronicles the Sister's journey from Seattle in 1902, to Nome, Fairbanks, and finally Anchorage. The program was produced by Mishler/Biddle Advertising and Design of Anchorage, in cooperation with the hospital's Community Relations and Media Services Departments.

History of Medicine in Alaska

JEAN COLESBERRY PERSONS, M.D.

Jean Persons, M.D., the daughter of Episcopal missionaries, has two birth certificates. She was born in Philadelphia on March 18, 1925. However her birth also was recorded in Ivy Depot, Virginia where her parents were stationed.

When Jean was two her parents were sent to the fishing village of LaGloria, Cuba where they were to spend the next six and one-half years. Cuba was to provide two memorable and potentially terrifying experiences.

First, there was the hurricane of 1932 which heavily hit their village. In the fierce winds, houses were blown down, and many people were injured or killed by the flying corrugated tin roofs. Jean remembers her house being used as a surgery where an amputated leg was buried and then dug up by her dog.

When the Batista revolution occurred, her father stayed behind. He sent his family home. But as their train left, the railroad station was blown up. Then, the evacuation plane following their own was shot down.

Jean's mother was to die of cancer, and Jean was sent to a boarding school run by Episcopal nuns in Peekskill, N.Y.. In the fall of 1943, she entered Louisiana State University in Baton Rouge. She was eager to finish school, carrying a heavy load and using correspondence courses to complete her degree. It was not an easy time, since she did not have enough money and often went to bed hungry.

Her father wanted her to be a doctor which was his own unfulfilled dream. Agreeing that this was a way to do what she wanted, Jean entered LSU medical school and was graduated in 1950. She served an internship at Charity Hospital, a 3,000 bed hospital with professors from Tulane and LSU. This was followed by a year's residency in internal medicine.

Seeking a different experience, she went west to Crownpoint, New Mexico to work on an Indian reservation. The transient Navajo lived in squalid quarters with minimal pay and contaminated water. Babies were tended by the smaller children, and consequently diarrhea was a constant and mostly fatal disease.

Jean recognized that the Indians trusted their Medicine Men. She invited them to the clinic and showed them how to use the otoscope and the stethescope. She felt honored when they showed her their skills.

The Bureau of Indian Affairs offered her a job at the new Native Service Hospital in Anchorage. Because the building was not completed, she was intrigued when she was asked to spend three months in Tanana.

She embarked for Alaska, stopping at Sitka where a dentist told her that what she needed to know most in the Bush was how to pull teeth. Then he taught her how.

Tanana had a 30 bed hospital, half of which was set aside for TB patients. There were five RNs and two LPNs. At all times the beds were full. There were occasions when there were as many as 70 in the hospital. Small children were doubled up by diagnosis or placed in cardboard boxes.

Jean's first patient was the man she discovered on her initial inspection of the hospital. She found a chair in the clinic covered with a sheet—underneath was a very cold and rigid man. Since most of the planes used to transport people between the villages were small, it had become the custom to sit the corpse so that as rigor mortis set in, the body would be in a sitting position. This way the pilot could put the body in the seat next to him and fly it home.

Shortly after arrival, Jean was put to test. A two-year old girl had been mauled by sled dogs. The child literally was scalped. Suturing the severed trachea, through which bloody air bubbled, and the sternocleidomastoid muscle required several hours. Fortunately the facial bites were minor. The child survived and much later when she was married was considered a beautiful bride. Happily for Jean, the pilot who had brought the child told everyone that the "new doc is okay."

Fr. Norman Elliott, Rector of All Saints' Episcopal church, who was then Archdeacon of the Yukon, tells of a man who dislocated his shoulder. Fr. Elliott found himself helping Dr. Persons. She read from an instruction manual while he pulled in the direction required. The shoulder was fixed. There were many such treatments learned on the spot.

The airplane was the ambulance of the Bush. Although there were many times when flying conditions were marginal, the choice was often between the doctor taking a chance or the patient having none. A pilot, Garfield Hansen offered to move to Tanana with his Piper supercruiser if Jean could use him. She could and did. His assistance allowed her to make many field trips to her villages.

She also went out on emergency calls. In the middle of a dark night, she received a call from Huslia reporting a child with 105°F fever and a stiff neck. Hansen took a look at the weather and, shrugging his shoulders, said "a 50-50 chance." They took off in the fog and could navigate only by following the Koyukuk River to Huslia. They had been assured that there would be lights on the landing field. There were—two lanterns at the same end of the short field. They made it and took the child with meningitis back to Tanana.

In January of 1954, Toughy Edgington, a bush pilot, travelling with his pregnant wife and an older Indian woman, crashed. Since planes should not fly in temperatures below 40°F, Jean took off by dog sled. She was careful to collect supplies, especially morphine and I.V. fluids, which she carried between her legs to keep them from freezing. As she departed, she was handed a revolver and reminded that there were wolves. At one point, her driver went through the ice but fortunately was unharmed.

After three hours, they reached a cabin and changed dog teams. Another three hours elapsed until they arrived at the crash site. It was at the bottom of a hill. Somehow the two women had gotten Toughy out of the badly wrecked plane and up the hill to a cabin. This undoubtedly saved his life. Toughy, well named, was in shock with a broken hand, legs, and jaw. Jean was able to stabilize him. Bob Byers had flown from Fairbanks, a masterful feat in the weather. With his help, they improvised a stretcher and carried Toughy down to Byers' plane.

Receiving a radio call from Nulato that a pregnant woman was bleeding, Jean started to the village. When she arrived, the bleeding had slowed down, and there were no contractions. Jean and her patient decided that it would be best to go to Tanana. Things were going well until the plane lost altitude, and the patient promptly went into labor. Hansen held the flashlight while a little girl was delivered. Jean could not find her scissors to cut the cord. Hansen lent her his pocket knife. Somehow the news of this was reported Outside. Jean received pocket knives from around the country, including one from the U.S. Surgeon General.

In the summer of 1953, Jean had gone to Anaktuvuk Pass. While it was July 4th, there was snow on the ground. She was amazed to see many goiters. The population was small, but the percentage of goiter high. The source of the villagers' fish was an inland lake, and Jean found that the trader brought only uniodized salt. As soon as the salt was iodized, the goiters began to disappear.

There were only about 50 adults in the village of Anaktuvuk. The year before there had been a measles epidemic. It had almost destroyed this fine tribe. Jean noticed that there were about 15 adult males and probably 30 women. Most of the women were pregnant, the practical Eskimo way of repopulating.

Jean did much dentistry. She remembers one village where she kept pulling teeth along with treating other complaints for many hours. At the end she counted 267 teeth in her bucket. After she reported this, a dentist was sent. Jean was grateful, because she had discovered that pulling and especially filling teeth was not her forte.

When Jean first went to Tanana, 140 miles west of Fairbanks, she considered it her territory. However, pilots would bring patients from distant villages, and she talked regularly by radio to others. She finally asked headquarters in Juneau what her territory was. She was told that it included any villages she talked to or from where patients came. Looking on a map, she discovered that she was serving from Beaver down river to Kaltag as well as all the Koyukuk villages and Anaktuvuk Pass.

In June of 1956, Jean married Bernardus J. Smit, a Dutchman, who was a trouble shooter for the Northern Commercial stores. The same year, she was transferred to Anchorage where she served as liaison between the Alaska Native Service and the professional community.

Two years later, when her husband was sent to Bethel, she worked full time at the hospital and clinic there. Bethel was bigger than Tanana and served some 75 villages. Her nurse–interpreter was outstanding. By now Jean had learned enough Yupik to start a conversation, but she depended upon her nurse for details. There were too many patients; sometimes 100 in an afternoon. It become a problem of separating measles from hepatitis. While this was not the best of medicine, it was better than none.

Jean was forced to cut back on her long hours by the arrival of Michele, January 1959, and then by Renee, November 1960. However, she continued to work four or five hours each day.

Her husband decided they would visit his home in the Netherlands. Here their youngest daughter, Tammy, was born. Afterwards they returned to California where Jean put her husband through law school.

In 1973, Jean divorced her husband and returned to Alaska. An anticipated position with the Alaska Native Service did not materialize, so she worked for Michael Bierne, M.D.. Ends did not always meet.

Life improved immeasurably when in 1976, Jean married Dr. Robert Whaley, M. D.. Currently, she practices with the Anchorage Medical and Surgical Clinic. The Whaleys enjoy scuba diving, flying, and an annual BMW motorcycle trip through the Alps. Dr. Jean Persons has come a long way from Cuba to Alaska to find an angle of repose.

Gwynneth Gminder Wilson
ASMA Auxiliary History Project

Thanks to Michele Whaley for lending papers written by and about Jean Persons and for discussing the material.

A **NEW** H_2 Antagonist

AXID® 300mg

nizatidine

Effective once-nightly
duodenal ulcer therapy available in a

Unique Convenience Pak

for better patient compliance

AXID®
nizatidine capsules

Brief Summary. Consult the package insert for prescribing information.

Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer In most patients, the ulcer will heal within four weeks.

Axid is indicated for maintenance therapy for duodenal ulcer patients, at a reduced dosage of 150 mg h s after healing of an active duodenal ulcer The consequences of continuous therapy with Axid for longer than one year are not known

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists

Precautions: *General*—1 Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy

2 Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done Part of the dose of nizatidine is metabolized in the liver In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects

Laboratory Tests—False-positive tests for urobilinogen with Multistix* may occur during therapy with nizatidine

Drug Interactions—No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system, therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b i d , was administered concurrently

Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect There was a dose related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high dose males compared to placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups The rate of hepatic carcinoma in the high dose animals was within the historical control limits seen for the strain of mice used The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement compared to concurrent controls, and evidence of mild liver injury (transaminase elevations) The occurrence of a marginal finding at high dose only in animals given an excessive, and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery is not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, and the mouse lymphoma assay

In a two-generation, perinatal and postnatal, fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose, and in Dutch Belted rabbits at doses up to 55 times the human dose, revealed no evidence of impaired fertility or teratogenic effect, but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers—Nizatidine is secreted and concentrated in the milk of lactating rats Pups reared by treated lactating rats had depressed growth rates Although no studies have been conducted in lactating women, nizatidine is assumed to be secreted in human milk, and caution should be exercised when nizatidine is administered to nursing mothers

Pediatric Use—Safety and effectiveness in children have not been established

Use in Elderly Patients—Ulcer healing rates in elderly patients are similar to those in younger age groups The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups Age alone may not be an important factor in the disposition of nizatidine Elderly patients may have reduced renal function

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo Among the more common adverse events in the domestic placebo-controlled trials, sweating (1% vs 0 2%), urticaria (0 5% vs <0 01%), and somnolence (2 4% vs 1 3%) were significantly more common in the nizatidine group A variety of less common events was also reported, it was not possible to determine whether these were caused by nizatidine

Hepatic—Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients possibly or probably related to nizatidine In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L), and in a single instance, SGPT was greater than 2,000 IU/L The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients All abnormalities were reversible after discontinuation of Axid

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo Rare reports of gynecomastia occurred

Hematologic—Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist On previous occasions, this patient had experienced thrombocytopenia while taking other drugs

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine than in placebo patients Rash and exfoliative dermatitis were also reported

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported

Overdosage: There is little clinical experience with overdosage of Axid in humans If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy Renal dialysis for four to six hours increased plasma clearance by approximately 84%

Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal Intravenous LD_{50} values in the rat and mouse were 301 mg/kg and 232 mg/kg respectively PV 2091 AMP [041288]

Axid* (nizatidine, Lilly)

Axid* (nizatidine, Lilly)

Lilly

Eli Lilly and Company
Indianapolis, Indiana
46285

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ARMY RESERVE MEDICAL PROFILE NO.7

ALLAN J. HAMILTON, M.D.

Neurosurgical Resident and Research Fellow, Massachusetts General Hospital, Boston, Massachusetts. Captain, U.S. Army Reserve.

EDUCATION Ithaca College, B.A. (Magna Cum Laude) Hamilton College (Pre-med); Harvard Medical School.

RESIDENCY General Surgical Internship. Neurosurgical Residency, Massachusetts General Hospital.

CONTINUING EDUCATION Neurology and Neuro surgery Research Fellowship Training, National Institutes of Health.

OUTSTANDING ACHIEVEMENTS Olsen Memoria Fellowship, National Masonic Medical Research Foundatio Albert Schweitzer Fellowship, International Albert Schweitz Foundation; Harvard Medical School Cabot Prize for Best Senior Thesis; recently published article, "Who Shall Live and Who Shall Die" in Newsweek Magazine.

"The work I'm doing in the Army Reserve fits perfectly with my academic research interests in civilian life. The Army is very concerned with the effects of high-altitude cerebral edema, which is a mirror model of cerebral hypoxia, something I deal with every day in our neurosurgical intensive care unit. I couldn't ask for a smoother transition. And that's true for a lot of Reserve physicians. All we really do is change our clothes not our mindset.

"Some of the projects the Army is undertaking are on the cutting edge of research. For example, I'm currently involved in developing for the Army a prototype of a non-invasive intracranial pressure-monitoring device that we hope will allow us to measure pressure changes as the brain swells—without drilling holes in the skull. If we can get our design to work, such a device could revolutionize high-altitude medicine as we as civilian neurosurgical care.

"The quality of medicine and the caliber of people I've been associated with in the Army Reserve are, without question, equal to civilian hospitals. In fact, I'm giving serious consideration to applying for an active duty academic position in Army Medicine when my residency ends at Massachusetts General."

Find out more about the medical opportunities in the Army Reserve. Call toll free 1-800-USA-ARMY

ARMY RESERVE MEDICINE.
BE ALL YOU CAN BE.

Soldier being examined for effects of high-altitude cerebral edema.

Glimpses of Alaskan Medical History

Edited by Robert Fortuine, M.D.

A Primer of Aleut Medicine

In 1785 Catherine II of Russia appointed an English captain, Joseph Billings, to head an expedition that was intended to increase geographical knowledge of the region between Asia and America. A German, Carl Heinrich Merck, was added to the command in 1786 as physician and naturalist. Following several years of exploration in Eastern Siberia, Billings finally sailed for Alaska in the spring of 1790. During May and June the ship cruised among the Aleutian Islands, spending a couple of weeks at Unalaska before moving on to Kodiak and Prince William Sound, and finally back to Kamchatka in October. The following year Billings and Merck explored the region around the Bering Strait and the Chukchi Peninsula.

Dr. Merck's detailed account of his journeys contain a wealth of information on the geography, birds, plants, sea mammals, and fish of the places he visited. Of even greater importance today are his observations on the Natives and their customs, many of which relate to health, disease, and the stages of life. Merck was in fact the first European to give a substantive account of Alaska Native traditional medicine, especially that of the Aleuts. Unlike his predecessors, he probably had the services of an experienced Aleut-Russian interpreter at Unalaska, where the Russians had already maintained a trading post for more than ten years.

The excerpts below are translated by Fritz Jaensch from a previously unpublished manuscript that was discovered in Germany in 1935.

"A man may not do any work with a hatchet while his wife is pregnant for fear that he might kill the child.

"While childbirth is in progress, the people take all male clothing, which hangs or lies about, off to another hut. Or they put all of it in a heap, which they cover with a straw mat. They do this because they believe that otherwise the men could come to harm because of the cleaning after the birth. . . . And everyone of male gender, both young and old, must leave the hut once the child is born. The women do not touch the child until after the afterbirth has been expelled. Then the mid-wives tie the navel cord with a tendon string and cut it off with a sharp piece of shell or with an arrowhead. This cutting instrument is then buried together with the afterbirth. The newborn child is first of all washed down with urine, then with cold, fresh water. Thereafter, one of the mid-wives takes the child to her bosom and warms it over a small straw fire.

"When everything is over, then all men, big and small, have to bathe in a brook or lake, even in winter. The mother is cared for and treated to clams and fresh fish for several days. But usually she goes after water again on the third day. If the mother is too weak to suckle the child, then the child must be satisfied with sucking a piece of seal's lard. A stick is pierced crossways through the piece of fat so that the child will not swallow it. . . .

"They know almost no cure against diseases, except bleeding *(Ukhtosh)*. Other than that, one might count the artifices of their shamans a cure. They trust in them very much; and the prophecies of the shamans are to the people a sure indication of their fate. . . .

"The symptoms of some of their diseases are a hardening of the inner organs. Such an organ feels like a stone. If a pain-inflicted part of the body seems favorable for bleeding, then a person experienced in this art will pierce obliquely, deep into the tissue. If plenty of blood flows, then they have good hopes for recovery. When this does not happen they fear the advent of death. Most of the times they bleed themselves between the ribs at the breast. If the heart seems to be sick *(Kanokh Nanalli)*, or if they suffer nausea and lack of appetite, then they scratch themselves below the tongue to bleed. They also warm sick places of the body with hot stones, wrapped in grass.

"Wounds of the venereal kind *(Shiakhsh)* have been known to them only since the arrival of the Russians. If a person is afflicted with this then they treat it in the same way they treat other wounds.

"Wounds are treated with boiled roots of Angelica, internally and externally. Internally they also give roots and leaves of sorrel, *Nenelli*. The vein where bleeding is done is called *Namiak*.

"They build a small cabin for the patient. Only a man, experienced in the treatment of the sick, stays with the patient. No woman is allowed to stay with the patient; first of all because they think that women are unclean. And furthermore they do not trust the looks of their eyes. They think that it gets worse with the patient because of such looks."

REFERENCE

Merck, Carl Heinrich. Siberia and northwestern America 1788-1792. The journal of Carl Heinrich Merck, naturalist with the Russian scientific expedition led by Captains Joseph Billings and Gavriil Sarychev. Ed. by Richard A. Pierce and trans. by Fritz Jaensch. Kingston, Ontario: Limestone Press, 1980. Pp. 175-176.

RESOLUTIONS

ADOPTED AT THE ALASKA STATE MEDICAL ASSOCIATION ANNUAL MEETING

Fairbanks, Alaska

JUNE 11, 1988

RESOLUTION 88-1

SUBJECT: Veterans Administration Hospital

WHEREAS, the current VA health care delivery system in Alaska has assured veterans access to quality health care services in their own communities, and

WHEREAS, Alaska has more than 71,000 veterans and approximately 52 percent of all veterans reside outside the Municipality of Anchorage, and

WHEREAS, there are significant uncertainties with the U.S. Corps of Engineers and Veterans Administration's cost savings estimates supporting their plans to construct VA beds at Elmendorf AFB, and

WHEREAS, Alaska's health care system is dependent on the participation of all population groups to maintain medical technologies and routine and specialized services currently available in Alaska, and

WHEREAS, any change in the health care delivery system would severely impact Alaska's already fragile delivery system, and

WHEREAS, VA administrators and planners have failed to take into account veterans' preference or health care, and

WHEREAS, the current system allows veterans to be cared for in their own home communities, and

WHEREAS, at a time of growing federal budgetary deficits, the federal government should be discouraged from contemplating construction of veterans' hospital beds in Alaska, and

WHEREAS, the proposal shifts the cost of health care to other payers and does not decrease the total cost of health care in Alaska; therefore

BE IT RESOLVED that the Alaska State Medical Association affirms its position that the Veterans Administration should not build an inpatient wing at Elmendorf AFB Hospital in Anchorage nor should any beds be utilized for inpatient care; and that veterans be allowed to continue to receive quality health care services from existing capacity in the private sector throughout Alaska.

RESOLUTION 88-2

SUBJECT: ASMA AIDS Policy

BE IT RESOLVED that the Alaska State Medical Association commends Alaskan physicians, nurses, and other health professionals in hospitals and other health institutions for providing competent and humane care to all patients, including patients critically ill with AIDS and AIDS-related conditions, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association urges health professionals who have primary responsibility for a patient's well being, in keeping with their professional ethics, to continue providing high-quality, non-judgmental care to their patients.

RESOLUTION 88-3

SUBJECT: Nuclear Test Ban

WHEREAS, a nuclear disaster would bring unimaginable suffering to the people of Alaska as well as the rest of humanity, and

WHEREAS, there is no present or foreseeable medical system capable of responding to a nuclear disaster, and

WHEREAS, testing is necessary for the development of nuclear weapons, and

WHEREAS, more and more persons in both the USA and the USSR are coming to appreciate the need for the control of nuclear weapons; therefore

BE IT RESOLVED that the Alaska State Medical Association recognizes the threat from nuclear weapons to the future health of the people of Alaska and supports the establishment of a mutual, verifiable, and comprehensive *Nuclear Test Ban.*

RESOLUTION 88-4
SUBJECT: Commending Rodman Wilson, M.D.

WHEREAS, Rodman Wilson, M.D., as the Chief Health Officer for the Municipality of Anchorage, worked diligently and faithfully to provide a smoke-free environment for all employees and to safeguard their health, and

WHEREAS, Rodman Wilson, M.D. overcame difficult and vociferous opposition and proceeded to oversee the passage of statutes that provided a smoke-free environment for the Municipality of Anchorage; therefore

BE IT RESOLVED that the Alaska State Medical Association commends Rodman Wilson, M.D., for his dedication and resolve in providing a healthier environment for the citizens of Anchorage.

RESOLUTION 88-5
SUBJECT: Supporting Study of Decriminalization of Marijuana Possession

WHEREAS, substance abuse has become an increasing concern to society in recent years, and

WHEREAS, there appears to be strong evidence to support the view that marijuana usage is a greater problem and concern than it was in the 1970s when the Alaska State Medical Association last studied this issue; therefore

BE IT RESOLVED that the Alaska State Medical Association study our position regarding decriminalization of marijuana possession in light of currently available evidence of the consequences of marijuana use by children and adults, both as a gateway drug into other substance abuse and as a drug with harmful effects.

RESOLUTION 88-6
SUBJECT: Calling for an Alaska Liability Task Force

WHEREAS, the problems with availability and affordability of liability insurance continue to diminish the availability of needed goods and services for Alaskans, and

WHEREAS, the legislature has been unable or unwilling to address these issues in any meaningful way, and

WHEREAS, the Lieutenant Governor at the 1987 ASMA annual meeting in Juneau proposed a dialogue among interested parties as a means of addressing this issue yet did nothing to initiate that dialogue; therefore

BE IT RESOLVED that the Alaska State Medical Association calls upon the governor to convene and fund a task force outside the legislative process to include trial lawyers, impacted professions and industries, the general public, and the insurance industry to study the issues and to make a series of recommendations to the sixteenth legislature by January 1989.

RESOLUTION 88-7
SUBJECT: Commending the American Lung Association of Alaska

WHEREAS, since 1968 the Alaska State Medical Association has condemned cigarette advertising and promotion, and

WHEREAS, the American Lung Association of Alaska is a volunteer organization with an interest in promoting improved personal health through many efforts aimed at Alaskan health professionals and patients, and

WHEREAS, the American Lung Association of Alaska worked effectively with the legislature in encouraging passage of Senate Bill 339, restricting tobacco access to minors; therefore

BE IT RESOLVED that the Alaska State Medical Association commends the American Lung Association of Alaska for their effective efforts in promoting personal health with their successful encouragement of legislation restricting tobacco access to minors.

RESOLUTION 88-9
SUBJECT: Supporting Revision of Alaska Statutes Regarding Alcohol Abuse

WHEREAS, alcohol abuse is a problem throughout Alaska, and

WHEREAS, current legislation regarding protective custody (A.S. 47.37.170) supports abuse of alcohol rather than discourages it, and

WHEREAS, current legislation concerning those incapacitated by alcohol (A.S. 47.37.180-200) is ineffectual and detrimental to the interests of those it purports to assist; therefore

BE IT RESOLVED that the Alaska State Medical Association seek legislation to revise appropriately the statutes regarding protective custody and treament of those incapacitated by alcohol, and

BE IT FURTHER RESOLVED that the lead premise for these changes in legislation be recognition of an individual person's responsibility for the social, family, legal and financial consequences of their disease of alcohol abuse.

RESOLUTION 88-10
SUBJECT: Increasing Alaska State Medical Association Political Activities

WHEREAS, onerous laws and regulations are threatening the quality of personal health care, and

WHEREAS, individual physicians acting alone are not able to impact the system significantly, and

WHEREAS, all bureaucrats are supported by appropriations enacted by legislators and signed by the executive; therefore

BE IT RESOLVED that the Alaska State Medical Association continue its involvement in the political process through maintaining an active lobbying presence in Juneau, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association, through the Alaska Medical Political Action Committee (ALPAC), continue efforts to support legislators responsive to medical issues and patients, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association support specific candidates for State House, State Senate, and Governor whose records and statements promote continued improvements in the quality and availability of medical care for Alaskans and continued improvements in public health.

RESOLUTION 88-11
SUBJECT: Impaired Physicians Program and Agreement

WHEREAS, this memorandum of understanding between the Alaska State Medical Board (the "Board"), the Alaska State Medical Association (the "Association"), and the Division of Occupational Licensing (the "Division") provides as follows:

WHEREAS, the Board has the statutory authority to discipline any physician licensed in Alaska when the physician uses intoxicating liquors, narcotics or other drugs to the extent that he is unfit for the performance of his professional obligations and duties, or when he is mentally or physically incompetent to practice his profession with safety to his/her patients and the public, and

WHEREAS, the Association has instituted a voluntary program whereby it will investigate or intervene with any physician in Alaska who by reason of illness, excessive use of alcohol, drugs, narcotics, chemicals or other type of material allegedly is unable to practice medicine with reasonable skill and attention to the safety of his/her patients (hereinafter referred to as "impaired physician") so as to facilitate getting the physician into treatment and to monitor his/her recovery, and

WHEREAS, the Board and the Association wish to coordinate their activities regarding impaired physicians to facilitate complementary performance of the disciplinary role of the Board with the rehabilitative role of the Association and to avoid duplication of effort and misunderstandings to the extent possible in cases of simultaneous involvement; therefore

BE IT RESOLVED that the Alaska State Medical Association agrees to a Memorandum of Understanding between the Alaska State Medical Association, Alaska State Medical Board, and Alaska Division of Occupational Licensing; for an Impaired Physician Treatment Program and Monitored Treatment Program Contract.

Note: Copies of the Memorandum of Understanding, Impaired Physicians Treatment Program, and Monitored Program Contract are available through the Alaska State Medical Association.

RESOLUTION 88-12
SUBJECT: Statewide Tumor Registry

WHEREAS, cancer is recognized in all communities as a health concern that has the potential to affect all individuals, and

WHEREAS, it is necessary in order to understand trends in any disease to have adequate vital statistics of occurrence rates and death rates by age, sex, race and other parameters, and

WHEREAS, there currently are several very active tumor registries in a few Alaskan communities but no statewide registry to compile and report cancer statistics, and

WHEREAS, there is already existing legislation providing for a statewide tumor registry; therefore

BE IT RESOLVED that the Alaska State Medical Association supports the concept of a statewide tumor registry, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association encourages the state legislature to fund such a registry during the next legislative session.

RESOLUTION 88-13
SUBJECT: Thanking Providence Hospital

WHEREAS, interest in medicine and health research in the arctic has never been higher and numerous opportunities exist to improve the health of all Alaskans, and

WHEREAS, crucial to improvement of health is excellence in scientific research and dissemination of findings to physicians and other health care professionals, and

WHEREAS, scientific publications are essential to enable peer review of research and prompt communication of important information, and

WHEREAS, scientific publications are rarely, if ever, economically self-supporting and *Alaska Medicine* is no exception, and

WHEREAS, Providence Hospital provided substantial financial support to *Alaska Medicine* and has pledged its future support; therefore

BE IT RESOLVED that the Alaska State Medical Association thanks Providence Hospital for its support of *Alaska Medicine.*

RESOLUTION 88-14
SUBJECT: Thanking Humana Hospital-Alaska

WHEREAS, interest in medicine and health research in the arctic has never been higher and numerous opportunities exist to improve the health of all Alaskans, and

WHEREAS, crucial to improvement of health is excellence in scientific research and dissemination of findings to physicians and other health care professionals, and

WHEREAS, scientific publications are essential to enable peer review of research and prompt communication of important information, and

WHEREAS, scientific publications are rarely, if ever, economically self-supporting and *Alaska Medicine* is no exception, and

WHEREAS, Humana Hospital-Alaska provided substantial financial support to *Alaska Medicine* and has pledged its future support; therefore

BE IT RESOLVED that the Alaska State Medical Association thanks Humana Hospital-Alaska for its support of *Alaska Medicine*, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association commends Humana Hospital-Alaska for its commitment to excellence in science, medicine and public health.

RESOLUTION 88-15
SUBJECT: AIDS Policy in Alaska

WHEREAS, the AIDS epidemic has resulted in the deaths of thousands of people and the virus, HIV, has infected hundreds of thousands of people, and

WHEREAS, scientists and researchers now believe that effective treatment, drugs and vaccines may not be available for ten years or more, making it essential that efforts to control this epidemic rely on education aimed at modifying or eliminating behaviors that place individuals at high risk of HIV infection, and

WHEREAS, Alaskans have responded to AIDS by developing a statewide program by appropriating state funds, and by implementing effective policies and programs; therefore

BE IT RESOLVED that the Alaska State Medical Association commends the governor, legislature, Senate Select Committee on AIDS, and the Department of Health and Social Services for their leadership in responding to the AIDS epidemic, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association calls upon the governor to adopt a state policy on AIDS that will:

- — insure accessibility and availability of appropriate services to all persons infected with HIV;
- — provide appropriate education and training to the general public, to all health care professionals, and to members of groups known to be at high risk for developing AIDS;
- — develop and provide public health measures designed to control the disease; and
- — respond to the AIDS epidemic based on the highest standard of scientific and ethical principle.

RESOLUTION 88-18
SUBJECT: Intrusion of Government into Health Care

BE IT RESOLVED that the Alaska State Medical Association opposes the current actions of the U.S. Department of Labor in issuing a compliance directive related to AIDS and health care workers, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association opposes the further intrusion of the U.S. Department of Labor into the health care setting as outlined in procedures contained in its compliance directive and proposed rulemaking.

RESOLUTION 88-19
SUBJECT: Thanking Governor Cowper

BE IT RESOLVED that the Alaska State Medical Association commends Governor Cowper and the Alaska Legislature for their vision, leadership, and wisdom in creating the Alaska Science and Technology Foundation and pledges its support to make the Foundation a success.

RESOLUTION 88-20
SUBJECT: Thanking the Exhibitors

BE IT RESOLVED that the Alaska State Medical Association expresses our sincere thanks to the exhibitors for their presence and financial support which added appreciably to our annual meeting, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association expresses our particular thanks to the A.H. Robins Company; Glaxo, Inc.; Medical Indemnity Corporation of Alaska; Medical Insurance Exchange of California; Schering; Upjohn; Winthrop Pharmaceuticals; and Wyeth-Ayerst Laboratories, for their further sponsorship of scientific speakers or convention activities.

RESOLUTION 88-21
SUBJECT: Thanking the Convention Committee and Convention Volunteers

BE IT RESOLVED that the Alaska State Medical Association expresses our sincere thanks to the convention committee and the many volunteers who have assisted us with our annual meeting; and

BE IT FURTHER RESOLVED that the Alaska State Medical Association expresses our very special appreciation to Gayle Thieman whose tireless enthusiasm and well-arranged activities made our time in Fairbanks both enjoyable and informative.

RESOLUTION 88-22
SUBJECT: Thanking the Speakers

BE IT RESOLVED that the Alaska State Medical Association expresses our sincere thanks to our speakers who have contributed their knowledge and experience through their informative, challenging, and entertaining presentations; and

BE IT FURTHER RESOLVED that the Alaska State Medical Association particularly thanks Charles Steiner, M.D., for his efforts in arranging, organizing, and expediting the scientific program for this annual meeting.

RESOLUTION 88-23
SUBJECT: Thanking the Staff

BE IT RESOLVED that the Alaska State Medical Association expresses our sincere appreciation to Jodi (back home in the ASMA office), Gayle, Verna, Ray, and "ASMA Staff Auxilian" Judy for their extraordinary efforts in arranging, coordinating, organizing and managing our annual meeting.

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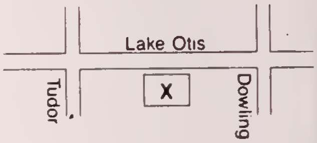

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News from the Alaska Public Health Association

RESOLUTIONS ADOPTED BY THE ALASKA PUBLIC HEALTH ASSOCIATION

MAY 1988

Resolution 88-1: Limiting Access to Tobacco

The Alaska Public Health Association,

Noting that the American Lung Association of Alaska estimates that a minimum of 1 in 7 people who died in Anchorage in 1986 died because they smoked cigarettes; and

Knowing that studies indicate that 90% of adults who smoke started smoking by the age of 19; and

Realizing that the age of 19 is, evidently, a critical age when social pressure lessens and concerns about health risks become more compelling; and

Finding that currently under state law tobacco can be sold to individuals 16 years of older; and

Finding that under existing statute there is some ambiguity whether smokeless tobacco is included in the age restriction law; and

Finding that presently there are no restrictions on the sale of tobacco from vending machines; and

Knowing that an important strategy for reducing tobacco's toll in Alaska is to lessen the number of Alaskan children who become addicted; therefore

Supports legislation which

1. increases the age for accessibility to all tobacco products to 19, and
2. makes it illegal to sell tobacco products from vending machines to persons under the age of 19; and

Recommends that the Association support SB 339 by:

1. sending letters to all members of the State Senate;
2. encouraging members to support this legislation through personal letters of support to their State Senators; and
3. urging individual members to encourage their respective organizations to send letters of support to all members of the State Senate.

Resolution 88-2: Establishment of a "User Fee" on Cigar, Pipe and Smokeless Tobacco

The Alaska Public Health Association,

Realizing that a study by the Indian Health Service found that 27.5% of all girls ages 5 through 18 years and 33.7% of all boys of that same age range report using smokeless tobacco products; and

Realizing that the same study showed that, on average, girls used 1.1 cans of snuff per week and boys used 1.4 cans per week; and that the average 18 year-old has been using smokeless tobacco for about 7.5 years; and that 16.9% of five-year old girls used smokeless tobacco; and

Knowing that the use of smokeless tobacco causes many serious health problems; most notably mouth cancer, throat cancer, gum disease, tooth loss, and slow healing of mouth wounds; and

Finding that a majority of states in the United States tax smokeless tobacco while Alaska does not—making smokeless tobacco relatively inexpensive in Alaska; and

Recognizing that studies show that a 10% increase in the cost of cigarettes leads to a 15% drop in teenage smoking and that similar results can be expected with increases in the price of smokeless tobacco; therefore

Supports legislation that creates users' fees and dedication of those revenues for health education and prevention; and

Recommends that the Association support HB 260 by:

1. sending letters to all members of the State Senate;
2. encouraging members to support this legislation through personal letters of support to their State Senators; and
3. urging individual members to encourage their respective organizations to send letters of support to all members of the State Senate.

Resolution 88-3: Earthquake and Tsunami Awareness

The Alaska Public Health Association,

Knowing that injuries are the leading cause of years of potential life lost and the leading cause of death in Alaska; and

Knowing that approximately half of the State of Alaska is considered seismically active with the most active region being the most populated region of the state in the wedge formed by lines running southeast and southwest with Fairbanks included at the apex; and

Knowing that in the twentieth century three of the top five most severe earthquakes in the world have been in Alaska:

- Ranked #5 was the 1957 Unalaska quake,
- Ranked #3 was the 1965 Rat Island quake,
- Ranked #2 was the 1964 Prince William Sound "Good Friday" quake; and

Knowing that just prior to this century in 1899 the 8.4 quake at Yakutat Bay elevated the coastline by 49 feet; and

Knowing that Alaska has sixty active volcanos along its portion of the Pacific "Ring of Fire" with the 1912 eruption of Mt. Katmai, 90 miles from Kodiak and 250 miles from Anchorage, having a discharge 24 times greater than that of the 1980 Mt. St. Helens eruption, and that only last year Mount Augustine threatened to knock out electrical power to the state's largest city and surrounding areas due to ash damaging the generators; and

Knowing that there have been more earthquakes worldwide and in Alaska in 1987 than in any year since 1976; and

Knowing that the strongest earthquake in the world in 1987 was a 7.6 Richter event in the Gulf of Alaska on November 30th; and

Knowing that a 7.2 Richter event was recorded in the Gulf of Alaska in March of 1988 which led to the third tsunami warning and resident evacuation of low lying areas in four months; and

Knowing that the February 6, 1988 earthquake that was centered close to Anchorage was strong enough to set off alarms at Pump Station Ten of the Trans-Alaska Oil Pipeline System in Isabell Pass in preparation of shutting the pipeline down; and

Knowing that "in any case we are satisfied that strain is accumulating in the Yakataga gap at a rate commensurate with the eventual occurrence of a great plate-margin earthquake" (1); and

Knowing that the Yakataga gap and Yakutat Bay are about equal distance between Juneau, the state's capital, and Anchorage, the state's largest city; and

Knowing that in 1964 90% of all Alaskan deaths from the earthquake were from tsunami; and

Knowing that there now are thousands more workers in larger coastal communities within the highly seismically active area of the state than there were in 1964; and

Knowing that "especially serious to all of the damaged communities is the loss of many fishing boats, seafood processing plants, and other waterfront installations which are the mainstay of their economy" (2); and

Knowing that hazardous materials (from acetic acid to zylene) exist throughout all of Alaska with most of them coming into the state and being initially stored in the very dock areas that are most threatened by earthquake and tsunami; and

Knowing that most earthquake related injuries, deaths, and building damage costs are due to non-structural damage, items which can often be secured or stabilized inexpensively with some awareness and education; and

Knowing that there are high density office buildings within yards of Holocene fault lines in Alaska (as in Anchorage); and

Knowing that most of the largest hotels and public buildings have no regular, obvious earthquake educational materials nor instructions for the people who use those facilities; and

Knowing that "a review of present preparedness in the state conducted in 1982-83 reveals that if a major earthquake were to occur today, the state and its major communities would be no better prepared than on March 27, 1964. Little planning for risk mitigation has been done. In fact, as a result of increased population and development that occurred in upper Cook Inlet, many scientists, planners, and administrators believe that another earthquake would have even greater impact on commerce and people than in 1964. Destruction of transportation systems and commerce in Anchorage, the major city and distribution center in the state, would affect the entire state economy" (3); and

Knowing that "unless the public increases its awareness and understanding of risk, and the mitigation management alternatives available, success at implementing hazard reduction programs is unlikely" (4); and

Knowing that "programs to reduce hazards and educate the public on preparedness in Mexico City were in need of greater support before the earthquake struck.... The importance and cost effectiveness of such programs constitute a lesson learned from this tragic event" (5); and

Understanding that there would have been many times the number of deaths in the great earthquake of 1964 had it not been for the fact that the earthquake struck at 5:36 P.M., on Good Friday, at low tide, with many people at home and away from their work places which were closed; and

Understanding that an earthquake in the cold, short days of late January, at high tide, at mid-work-week, at mid-work-day, would be devastating; therefore

Recommends that in order to reduce potential injury, reduce potential productive years of life lost, and reduce economic hardship and property loss, that the State of Alaska through its Governor and Legislature allocate sufficient funds and authorize the appropriate departments to:

1. Obtain or develop Alaskan specific earthquake and tsunami educational materials and programs to be made available through employers, using business as a common point clearing house, so that they may instruct their employees on how to react in the event of an earthquake occurring while they are at their job site; and

2. Obtain or develop Alaskan specific earthquake and tsunami educational materials and programs to be made available through employers, using business as a common point clearing house, so that they may provide materials to their employees on how to react in the event of an earthquake occurring when they are at home; and

3. Obtain or develop materials that have practical suggestions and recommended building standards for non-structural as well as structural items; and

4. Provide an educational environment for employers and employees, and communities in general to decrease the likelihood of unintentional earthquake related injuries, through the use of a statewide community-based effort to disseminate the developed materials and practical suggestions on earthquake safety mitigation strategies, provide seminars, press releases, and coordination support for community based groups to be developed and in place to tie into the various activities that will be occurring to remember the 25th anniversary of the 1964 Great Alaska Earthquake on 27 March 1989 and then for use on into the future; and

Recommends that in keeping with the lead set by California, and in a similar vein as October being "Fire Prevention Month" with many public awareness programs both in the schools and in the media, that the Governor and the Legislature officially designate every April as "Earthquake Preparedness Month."

REFERENCES

1. J.C. Savage, etal, "Strain Accumulation in the Shumagin and Yakataga Seismic Gaps, Alaska," 7 February 1986, "Science," page 587.
2. 1964 Geological Survey Circular 491, "A Preliminary Geologic Evaluation," page 25.
3. L. Selkregg, "The Great Alaska Earthquake 1964—Revisited," page 1.
4. L. Selkregg, 1983, "The Day the Earth Shook—Shock and After Shock."
5. Earthquake Engineering Research Institute, "Impressions of the Guerrero-Michoacan, Mexico Earthquake of 19 September 1985."

Resolution 88-4: Opposition to Plutonium Shipments Under a Proposed United States Agreement with Japan

The Alaska Public Health Association,

Noting that plutonium oxide powder is an extremely toxic, easily airborne nuclear fuel that can be lethal in minute amounts when ingested; and

Recognizing that the United States and Japan have recently concluded the negotiation of a 30-year nuclear cooperation agreement that would permit the shipping of plutonium by air over a polar route between Europe and Japan (1); and

Noting that the signing of the agreement would substitute a blanket 30-year approval for the present case-by-case review process that gives the United States ongoing control over the safety and security of plutonium shipments; and

Noting that it is predicted as much as 40 metric tons of plutonium oxide powder would be shipped from Europe to Japan every two weeks for several years starting in early 1990s; and

Noting that the treaty requires only that International Atomic Energy Agency standards are to be used, and these standards require a crash test velocity of only 44 feet/second, while USNRC standards require a velocity of 422 feet/second (l); and

Noting that a cask for safely shipping large quantities of plutonium by air has not yet been designed, and it is unclear whether it is possible to build a cask that can survive an airport crash and is large enough to ship the quantities of plutonium contemplated by the agreement; and

Noting that an environmental impact statement has not been prepared for the proposed agreement, although there is court precedent for the proposition that the movement of nuclear fuel qualifies as a major federal action requiring preparation of an impact statement; and

Realizing that in the event of a terrorist attack, in-flight mechanical failure, in-flight collision, crash on landing or take-off, the public health threat to the people of the United States and other countries exposed to hundreds of pounds of liberated powdered plutonium oxide could be catastrophic; and

Noting that the suit filed by the state of Alaska against the federal government on October 1, 1987 indicating that the agreement is illegal because it fails to take proper safeguards to protect the public health and environment, on October 3, 1987 was denied by the federal judge who refused to put an immediate halt to the consideration by the Department of Energy (USDOE) of the treaty with Japan; and

Noting further that the USDOE hurriedly pushed this agreement through to President Reagan's desk; and

Recognizing that both the House and Senate Foreign Relations Committee have notified the President that he should withdraw and renegotiate a new U.S./Japan Nuclear Cooperative Agreement because it does not meet the requirements of U.S. Nuclear Proliferation Law, therefore

> Opposes ratification of the nuclear cooperation treaty between Japan and the United States; and
>
> Will send copies of this resolution to the Alaska Congressional Delegation, the Governor, members of the state legislature, President Reagan, and the local media.

REFERENCES

1. Paul Leventhal etal. Air Transport of Plutonium Obtained by the Japanese from Nuclear Fuel Supplied by the United States, Nuclear Control Institute: Washington, D.C., March 3, 1987; House of Joint Resolution No. 35, in the Legislature of the State of Alaska, Fifteenth Legislature – First Session, Relating to the use of state airports for plutonium shipments under a proposed United States agreement with Japan.

Resolution 88-5: Expanding Eligibility for Prenatal Patients

The Alaska Public Health Association,

Knowing that early and complete prenatal care is critical in promoting positive pregnancy outcomes; and

Recognizing that the current eligibility policies relating to Medicaid benefits for low-income pregnant women often limit their access to prenatal care; and

Believing that low-income women are often at greater risk for problems during pregnancy that adversely affect their infants; and

Knowing that these adverse affects can potentially impact the health, development, and productivity of those infants throughout their lives, at great personal cost and cost to society; therefore

Recommends that Medicaid be expanded to increase the number of women eligible for prenatal care through redefinition of poverty level, extension of length of coverage, both prenatally and post-natally, and a single eligibility determination during pregnancy.

Resolution 88-6: Maintenance of Immunization Statutes in State of Alaska

The Alaska Public Health Association,

Knowing that the benefits of immunization outweigh its potential risks; and

Recognizing that the current Alaska immunization program has been effective in preventing immunizable diseases in Alaska during the past ten years; and

Understanding that high levels of immunity among the general population are necessary in order to minimize the possibility of transmission of vaccine-preventable diseases; and

Realizing that making the immunization of children a matter of parental choice will have the result that fewer children will be immunized and that the general level of immunity in the state will be lowered; and

Recalling that the House of Delegates of the American Medical Association has recommended that exemptions to vaccination requirements based on religious or philosophic beliefs should be repealed in states which have such statutory exemptions from mandatory immunizations; and

Knowing that the National Childhood Vaccine Injury Act of 1986 provides a mechanism by which those persons who sustain serious vaccine-related adverse events will be compensated without the need for litigation; and

Noting that regulations requiring the reporting of severe vaccine- associated adverse events by health care providers to the Alaska Division of Public Health already exist; and

Understanding that House Bill No. 277, which provides for a philosophical belief exemption to immunization and sets forth requirements for reporting of vaccine-related adverse events, has been introduced in the Alaska House of Representatives; therefore

> Reaffirms support for the continual use of the current immunization laws;
>
> Recommends opposition to House Bill No. 277 by letters from the Association to all members of the Alaska Legislature; and
>
> Urges the membership of the Association to oppose this legislation by personal letters to their legislators.

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My Turn

Winning the War and Losing the Peace

Passage of legislation to establish an Alaska Science and Technology Foundation and its approval by the governor is a great victory for research in Alaska. Applied research supported by that foundation can play a leading role in diversifying the state's economy. Basic research will create new knowledge and that too is essential for the long-term future of the state and the North. Victory, however, can quickly change to defeat if great care is not exercised in organizing, staffing, and operating this foundation.

The fiscal note of $6,000,000 to fund the foundation and its work in the coming biennium is adequate for a new program and body. It can provide a small staff, the preparation of priorities and processes, and support for several well-considered projects. That sum could also be used quickly to fund a large number of suggestions that always arise to spend money. Such an approach is tempting in a tight economy where many of our fine minds with good ideas are hard pressed. History should cause us to pause before we fall to temptation.

The Alaska Energy Center started in 1980 with great possibilities, good intentions, a deluge of suggestions, and considerable cash. Soon the money was gone and so too was public confidence and legislative support. Good work got lost in questions of intent and relevance. In a few months the Center was terminated; a sad end for a mechanism that could have supported science and technology.

The long-term success of the foundation depends not only on the experience and outlook of the board and staff but also on how well those two groups interact with the Alaska public, industry, government, and academia. The forming Act is very helpful in this regard, because it calls for a board of diverse experience. Further, the governor has solicited suggestions from the scientific and technical communities as well as the general public; that is a positive step in directing the conduct of scientific and engineering research to public purposes. There have been occasions when the emphasis has been otherwise or on what public policy might do specifically to help science and technology per se.

A crucial step in the process of organizing an effective foundation will be the selection of an executive director. It is this individual who will recruit a small supporting staff and that group will implement the directions of the board and otherwise carry out the mandates of the Act. Who the executive director is, his experience and his outlook, will quite necessarily affect how the foundation performs and how it relates to its constituencies.

Beyond giving the board the authority to select and employ an executive director, the Act is silent, as it should be.

Perhaps a list of questions can best describe the issues confronting the board in this task. Some that doubtlessly will be addressed follow:

- How important is a working knowledge of Alaska? Is an understanding of its people, economic condition, and methods of operation crucial—or will foundation experience elsewhere suffice?
- Should the executive director have a scientific and/or technical background? If so, is an applied or basic research background most desirable?
- Will a management specialist versed in governmental procedures and budget development be the person to start the foundation on its course of supporting research in Alaska?
- Is a Ph.D. degree necessary or even desirable for the executive director or is a range of scientific or technical experience with a B.S. or M.S. better fitted to respond to the many different problems which will face this individual?

It is important to move quickly but also carefully in appointing the board, hiring an executive director and other staff, and establishing a mode of operation. Spending money on projects should come later—after a specific direction has been set. A key element of success will be defining goals and objectives for funding basic research, applied research, and technological innovation and application. A common direction that the governor, the board, the staff, and the public are enthusiastic about will help ensure winning the peace.

Lyle D. Perrigo

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Auxiliary News

Alaska State Medical Association

The ASMA annual meeting is a time for spouses from all over the state to meet one another. This year they also met the new AMA Auxiliary President, Mary Strauss, from Maryland. Her message to the auxiliary seemed to hold a personal note for everyone there and to inspire each of us.

She spoke of the auxiliary not only as a community support organization but also as a self support group. In this age of high stress in the medical community, one of the most valuable benefits of the auxiliary is the friendship with other auxilians who understand the stresses which we face.

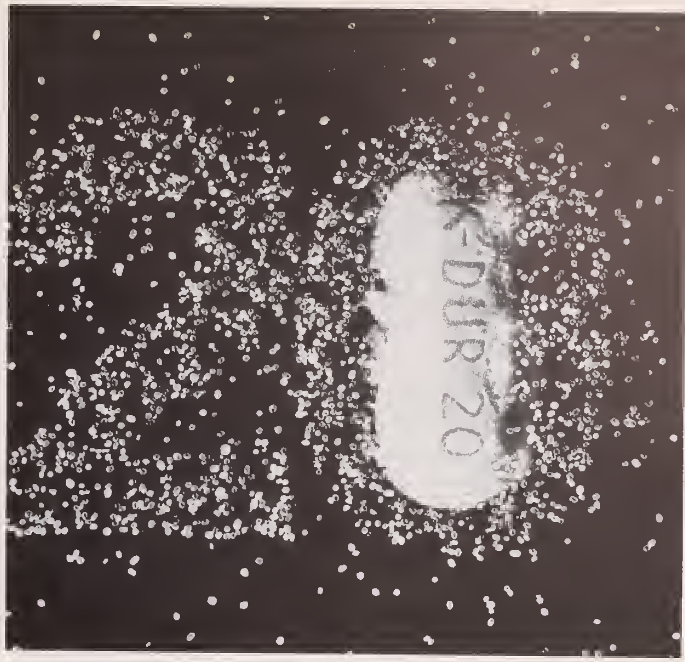
Cassie Hill, state president 1987-88, informed the auxilians of the work being done in tort reform. She also reported on the grass roots movement that is involving physicians and spouses at the precinct level in the Republican and Democratic parties. Cassie is to be congratulated on her accomplishments this past year as state president.

Maria Rundquist from Fairbanks is working toward revitalizing the Fairbanks Medical Society Auxiliary. An initial meeting and workshop is being planned in September. We would also like to see an auxiliary formed in the Kenai-Soldotna area.

I would like to hear from physician spouses throughout the state this year. It doesn't matter if you are a member of an organized auxiliary, the state auxiliary may still have something to offer you.

I am looking forward to working with all of you this coming year.

Alice Samuelson
ASMA Auxiliary President



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K-DUR™ Microburst Release System®
(potassium chloride) Sustained Release Tablets

INDICATIONS AND USAGE: BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS

1. For therapeutic use in patients with hypokalemia with or without metabolic alkalosis, in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.

2. For the prevention of potassium depletion when the dietary intake is inadequate in the following conditions: Patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis with ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy, and with certain diarrheal states.

3 The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated.

CONTRAINDICATIONS: Potassium supplements are contraindicated in patients with hyperkalemia since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: Chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g , spironolactone, triamterene).

Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium.

All solid dosage forms of potassium chloride supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation

WARNINGS: Hyperkalemia—In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment

Interaction with Potassium Sparing Diuretics—Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e g , spironolactone or triamterene) since the simultaneous administration of these agents can produce severe hyperkalemia

Gastrointestinal Lesions—Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage or perforation.

K-DUR tablets contain micro-crystalloids which disperse upon disintegration of the tablet These micro-crystalloids are formulated to provide a controlled release of potassium chloride The dispersibility of the micro-crystalloids and the controlled release of ions from them are intended to minimize the possibility of a high local concentration near the gastrointestinal mucosa and the ability of the KCl to cause stenosis or ulceration. Other means of accomplishing this (e g , incorporation of potassium chloride into a wax matrix) have reduced the frequency of such lesions to less than one per 100,000 patient years (compared to 40–50 per 100,000 patient years with enteric-coated potassium chloride) but have not eliminated them. The frequency of GI lesions with K-DUR tablets is, at present, unknown. K-DUR tablets should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis—Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, potassium acetate, or potassium gluconate.

PRECAUTIONS: The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis per se can produce hypokalemia in the absence of a deficit in total body potassium while acute acidosis per se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease, or acidosis requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the electrocardiogram, and the clinical status of the patient

Laboratory Tests: Regular serum potassium determinations are recommended In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis

Drug Interactions: Potassium-sparing diuretics; see **WARNINGS.**

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term carcinogenicity studies in animals have not been performed.

Pregnancy Category C: Animal reproduction studies have not been conducted with K-DUR. It is also not known whether K-DUR can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity K-DUR should be given to a pregnant woman only if clearly needed.

Nursing Mothers: The normal potassium ion content of human milk is about 13 mEq per liter. Since oral potassium becomes part of the body potassium pool, so long as body potassium is not excessive, the contribution of potassium chloride supplementation should have little or no effect on the level in human milk.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS,** and **OVERDOSAGE).** There have also been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS);** other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea. These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely

OVERDOSAGE: The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic electrocardiographic changes (peaking of T-waves, loss of P-waves, depression of S-T segment, and prolongation of the QT-interval). Late manifestations include muscle-paralysis and cardiovascular collapse from cardiac arrest.

Treatment measures for hyperkalemia include the following:

1. Elimination of foods and medications containing potassium and of potassium-sparing diuretics.
2. Intravenous administration of 300 to 500 ml/hr of 10% dextrose solution containing 10–20 units of insulin per 1,000 ml.
3. Correction of acidosis, if present, with intravenous sodium bicarbonate.
4 Use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia, it should be recalled that in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

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Letters to the Editor

Dear Editor,

Knowing of your interest in the childhood immunization program, I thought you'd like an update on the progress we've made toward addressing funding needs in FY'89.

Although the Administration's budget request is a slight increase over the FY'88 appropriation, for which I am glad, it still falls short of the amount necessary to adequately maintain the program. For this reason I've asked Senator Chiles, Chairman, and Senator Weicker, Ranking Republican Member, of the Labor, HHS and Education Appropriations Subcommittee, to provide additional funding for the childhood immunization program. I recommended that $136.62 million be appropriated for basic program needs, including funds to help cover the costs associated with the enactment of the Vaccine Compensation Amendments. I also recommended an $80 million appropriation to cover the amount authorized for the retroactive vaccine injury cases.

Childhood immunization is one of the most successful and cost-effective preventive health programs in this country. I'll monitor this issue carefully as we continue our work on the FY'89 Appropriation bills.

Ted Stevens
United States Senate

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Editorial

Last year the Council of the Alaska State Medical Association approved an ambitious, five year plan to establish *Alaska Medicine* as an outstanding scientific journal—a journal that would become mandatory reading for all those interested in health in Alaska and in the arctic. I believe that we are well on the way to reaching our goal.

During the past year, we have devoted most of our energies to rebuilding the journal. The journal was redesigned and reformatted. We hired a new publisher to typeset and print the journal; developed a completely new set of written policies and procedures to cover all aspects of production and publication; and began to do all copy editing in house by using an IBM computer and electronically transmitting only finished copy to the typesetter. The layout was reviewed, then changed, and a new and distinctive cover was designed. To give the journal a more professional look and to improve the appearance of insert advertisements, we changed to perfect binding.

While these formidable tasks were underway, we increased the number of issues from quarterly (4) to bimonthly (6). We were also able to increase our circulation from 800 to 1300 per issue.

Of far greater importance, we instituted strict peer review of all original contributed articles. In addition, we added several new features, including news from the Alaska Public Health Association and American Society for Circumpolar Health. Special articles were contributed by our major donors—the articles contributed were excellent and produced many favorable comments.

We also broadened our readership by extending invitations that, I am happy to report, were accepted to two professional health organizations—the Alaska Public Health Association and American Society for Circumpolar Health—to adopt *Alaska Medicine* as their official scientific publication.

Finally, I take great delight in reporting the following item. Last fall, we elected to submit *Alaska Medicine* to a national medical journal competition, one that is sponsored each year by Sandoz Pharmaceuticals. Although it was our first entry, *Alaska Medicine* was awarded an Honorable Mention Award for outstanding design and editorial qualities.

None of these activities or accomplishments would have been possible without the financial support of Providence Hospital and Humana Hospital-Alaska. Both hospitals have indicated that they will continue to support the journal and the Alaska State Medical Association in the future. In 1987 the journal operated in the black! With the continued support of our major donors and with the increasing advertising revenue due to our increased circulation and prestige, we will continue to do so.

We are pleased with the increasing prestige, circulation, and influence of the journal. The abstract of one *Alaska Medicine* article was reprinted by the Journal of the American Medical Association (JAMA). Several articles and editorials published in *Alaska Medicine* resulted in widespread media coverage. One article, in fact, was turned into a major, full page story by the Los Angeles Times.

Perhaps of more importance to Alaskans, the journal is increasingly read by our local, state, and federal governmental officials and policy makers. We are receiving requests for copies of journal issues dealing with the U.S. Arctic Research and Policy Act of 1984 and its implementation, with efforts to improve arctic health research funding and activities, with activities related to international cooperation on health topics among circumpolar countries, and with problems we are facing in providing health services in Alaska.

The future of the journal looks bright. *Alaska Medicine* represents an investment in Alaska and in our future. With adequate funds and our continued commitment to excellence in medicine, public health, and health research, we can make a permanent contribution to the health of all Alaskans. *Alaska Medicine* is rapidly becoming an important forum to address issues of critical importance to the health of Alaskans and will provide a permanent record of our quest for excellence.

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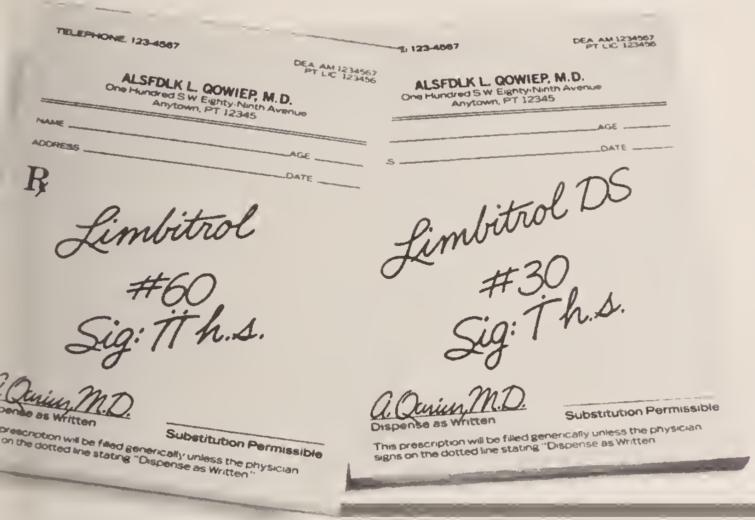

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Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants; concomitant use with MAOIs or within 14 days of monoamine oxidase inhibitors (then initiate cautiously, gradually increasing dosage until optimal response is achieved); during acute recovery phase following myocardial infarction.

Warnings: Use with caution in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur when used with anticholinergics. Closely supervise cardiovascular patients. Arrhythmias, sinus tachycardia, prolongation of conduction time, myocardial infarction and stroke reported with tricyclic antidepressants, especially in high doses. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations. Consider possibility of pregnancy when instituting therapy.**

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: Use cautiously in patients with a history of seizures, in hyperthyroid patients, those on thyroid medication, patients with impaired renal or hepatic function. Because of suicidal ideation in depressed patients, do not permit easy access to large quantities of drug. Periodic liver function tests and blood counts recommended during prolonged treatment. Amitriptyline may block action of guanethidine or similar antihypertensives. When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady-state concentrations of the tricyclic drugs. Use of Limbitrol with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Should not be taken during the nursing period or by children under 12. In elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: Most frequent: drowsiness, dry mouth, constipation, blurred vision, dizziness, bloating. Less frequent: vivid dreams, impotence, tremor, confusion, nasal congestion. Rare: granulocytopenia, jaundice, hepatic dysfunction. Others: many symptoms associated with depression including anorexia, fatigue, weakness, restlessness, lethargy.
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Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. Withdrawal symptoms also reported with abrupt amitriptyline discontinuation. Therefore, after extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

Overdosage: Immediately hospitalize patient. Treat symptomatically and supportively. I.V. administration of 1 to 3 mg physostigmine salicylate may reverse symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

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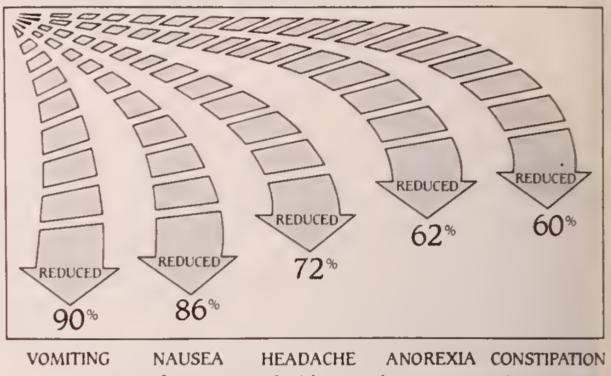

*Patients often presented with more than one somatic symptom.

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Skin and Skin Structure Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Proteus mirabilis, Proteus vulgaris, Providencia stuartii, Morganella morganii, Citrobacter freundii, Pseudomonas aeruginosa, Staphylococcus aureus* (penicillinase and nonpenicillinase-producing strains), *Staphylococcus epidermidis*, and *Streptococcus pyogenes*

Bone and Joint Infections caused by *Enterobacter cloacae, Serratia marcescens*, and *Pseudomonas aeruginosa*

Urinary Tract Infections caused by *Escherichia coli, Klebsiella pneumoniae, Enterobacter cloacae, Serratia marcescens, Proteus mirabilis, Providencia rettgeri, Morganella morganii, Citrobacter diversus, Citrobacter freundii, Pseudomonas aeruginosa, Staphylococcus epidermidis*, and *Streptococcus faecalis*

Infectious Diarrhea caused by *Escherichia coli* (enterotoxigenic strains), *Campylobacter jejuni, Shigella flexneri*,* and *Shigella sonnei** when antibacterial therapy is indicated

*Efficacy for this organism in this organ system was studied in fewer than 10 infections

Appropriate culture and susceptibility tests should be performed before treatment in order to isolate and identify organisms causing infection and to determine their susceptibility to ciprofloxacin Therapy with Cipro® may be initiated before results of these tests are known, once results become available appropriate therapy should be continued As with other drugs, some strains of *Pseudomonas aeruginosa* may develop resistance fairly rapidly during treatment with ciprofloxacin Culture and susceptibility testing performed periodically during therapy will provide information not only on the therapeutic effect of the antimicrobial agent but also on the possible emergence of bacterial resistance

CONTRAINDICATIONS

A history of hypersensitivity to ciprofloxacin is a contraindication to its use A history of hypersensitivity to other quinolones may also contraindicate the use of ciprofloxacin

WARNINGS

CIPROFLOXACIN SHOULD NOT BE USED IN CHILDREN OR PREGNANT WOMEN The oral administration of ciprofloxacin caused lameness in immature dogs Histopathological examination of the weight-bearing joints of these dogs revealed permanent lesions of the cartilage. Related drugs such as nalidixic acid, cinoxacin, and norfloxacin also produced erosions of cartilage of weight-bearing joints and other signs of arthropathy in immature animals of various species (SEE ANIMAL PHARMACOLOGY SECTION IN FULL PRESCRIBING INFORMATION)

PRECAUTIONS

General

As with other quinolones, ciprofloxacin may cause central nervous system (CNS) stimulation, which may lead to tremor, restlessness, lightheadedness, confusion, and very rarely to hallucinations or convulsive seizures Therefore, ciprofloxacin should be used with caution in patients with known or suspected CNS disorders, such as severe cerebral arteriosclerosis or epilepsy, or other factors which predispose to seizures (SEE ADVERSE REACTIONS).

Crystals of ciprofloxacin have been observed rarely in the urine of human subjects but more frequently in the urine of laboratory animals. Crystalluria related to ciprofloxacin has been reported only rarely in man, because human urine is usually acidic Patients receiving ciprofloxacin should be well hydrated, and alkalinity of the urine should be avoided. The recommended daily dose should not be exceeded Alteration of the dosage regimen is necessary for patients with impairment of renal function (SEE DOSAGE AND ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION)

Drug Interactions.

Concurrent administration of ciprofloxacin with theophylline may lead to elevated plasma concentrations of theophylline and prolongation of its elimination half-life This may result in increased risk of theophylline-related adverse reactions If concomitant use cannot be avoided, plasma levels of theophylline should be monitored and dosage adjustments made as appropriate.

Antacids containing magnesium hydroxide or aluminum hydroxide may interfere with the absorption of ciprofloxacin, resulting in serum and urine levels lower than desired, concurrent administration of these agents with ciprofloxacin should be avoided

Probenecid interferes with the renal tubular secretion of ciprofloxacin and produces an increase in the level of ciprofloxacin in the serum. This should be considered if patients are receiving both drugs concomitantly

As with other broad-spectrum antibiotics, prolonged use of ciprofloxacin may result in overgrowth of nonsusceptible organisms. Repeated evaluation of the patient's condition and microbial susceptibility testing is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Information for Patients

Patients should be advised that ciprofloxacin may be taken with or without meals The preferred time of dosing is two hours after a meal Patients should also be advised to drink fluids liberally and not take antacids containing magnesium or aluminum concomitantly or within two hours after dosing Ciprofloxacin may cause dizziness or lightheadedness, therefore patients should know how they react to this drug before they operate an automobile or machinery or engage in activities requiring mental alertness or coordination.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Eight *in vitro* mutagenicity tests have been conducted with ciprofloxacin and the test results are listed below

- Salmonella/Microsome Test (Negative)
- *E coli* DNA Repair Assay (Negative)
- Mouse Lymphoma Cell Forward Mutation Assay (Positive)
- Chinese Hamster V_{79} Cell HGPRT Test (Negative)
- Syrian Hamster Embryo Cell Transformation Assay (Negative)
- *Saccharomyces cerevisiae* Point Mutation Assay (Negative)
- *Saccharomyces cerevisiae* Mitotic Crossover and Gene Conversion Assay (Negative)
- Rat Hepatocyte DNA Repair Assay (Positive)

Thus, two of the eight tests were positive, but the following three *in vivo* test systems gave negative results.

- Rat Hepatocyte DNA Repair Assay
- Micronucleus Test (Mice)
- Dominant Lethal Test (Mice)

Long-term carcinogenicity studies in animals have not yet been completed

Pregnancy – Pregnancy Category C.

Reproduction studies have been performed in rats and mice at doses up to six times the usual daily human dose and have revealed no evidence of impaired fertility or harm to the fetus due to ciprofloxacin In rabbits, as with most antimicrobial agents, ciprofloxacin (30 and 100 mg/kg orally) produced gastrointestinal disturbances resulting in maternal weight loss and an increased incidence of abortion. No teratogenicity was observed at either dose After intravenous administration, at doses up to 20 mg/kg, no maternal toxicity was produced, and no embryotoxicity or teratogenicity was observed There are, however, no adequate and well-controlled studies in pregnant women SINCE CIPROFLOXACIN, LIKE OTHER DRUGS IN ITS CLASS, CAUSES ARTHF IMMATURE ANIMALS, IT SHOULD NOT BE USED IN PREGNANT WOMEN (SEE WARNINGS)

Nursing Mothers

It is not known whether ciprofloxacin is excreted in human milk, however, it is known that cip excreted in the milk of lactating rats and that other drugs of this class are excreted in human milk Bec and because of the potential for serious adverse reactions from ciprofloxacin in nursing infants, a dec be made to discontinue nursing or to discontinue the drug, taking into account the importance of th mother

Pediatric Use

Ciprofloxacin should not be used in children because it causes arthropathy in immature a WARNINGS)

ADVERSE REACTIONS

Ciprofloxacin is generally well tolerated During clinical investigation, 2,799 patients received 2,8 the drug Adverse events that were considered likely to be drug related occurred in 7 3% of cour related in 9 2%, and remotely related in 3 0% Ciprofloxacin was discontinued because of an adv 3 5% of courses, primarily involving the gastrointestinal system (1 5%), skin (0 6%), and central ne (0 4%)

The most frequently reported events, drug related or not, were nausea (5 2%), diarrhea (2 3 (2 0%), abdominal pain/discomfort (1 7%), headache (1 2%), restlessness (1 1%), and rash (1 1%)

Additional events that occurred in less than 1% of ciprofloxacin courses are listed below Tho quinolones are italicized

GASTROINTESTINAL *(See above)*, painful oral mucosa, oral candidiasis, dysphagia, intestina gastrointestinal bleeding

CENTRAL NERVOUS SYSTEM *(See above), dizziness, lightheadedness, insomnia, nightmare tions, manic reaction, irritability, tremor,* ataxia, *convulsive seizures, lethargy, drowsines malaise, anorexia, phobia, depersonalization, depression, paresthesia*

SKIN/HYPERSENSITIVITY *(See above), pruritus, urticaria, photosensitivity, flushing*, *angioedema, edema of the face, neck, lips, conjunctivae or hands*, cutaneous candidiasis, hy tion, erythema nodosum

SPECIAL SENSES. *blurred vision, disturbed vision, (change in color perception, overbrightne decreased visual acuity, diplopia, eye pain, tinnitus, bad taste*

MUSCULOSKELETAL *joint or back pain, joint stiffness*, achiness, neck or chest pain, flare-u

RENAL/UROGENITAL. *interstitial* nephritis, *renal failure*, polyuria, urinary retention, ureth vaginitis, acidosis

CARDIOVASCULAR. palpitations, atrial flutter, ventricular ectopy, syncope, hypertension, an myocardial infarction, cardiopulmonary arrest, cerebral thrombosis

RESPIRATORY epistaxis, laryngeal or pulmonary edema, hiccough, hemoptysis, dyspnea, b pulmonary embolism

Most of these events were described as only mild or moderate in severity, abated soon after discontinued, and required no treatment

In several instances, nausea, vomiting, tremor, restlessness, agitation, or palpitations we investigators to be related to elevated plasma levels of theophylline possibly as a result of a drug in ciprofloxacin

Adverse Laboratory Changes Changes in laboratory parameters listed as adverse events without relationship

Hepatic – Elevations of ALT (SGPT) (1.9%), AST (SGOT) (1 7%), alkaline phosphatase (0 8%) serum bilirubin (0 3%)

Hematologic – eosinophilia (0.6%), leukopenia (0 4%), decreased blood platelets (0 1%), e platelets (0.1%), pancytopenia (0.1%).

Renal – Elevations of. Serum creatinine (1 1%), BUN (0 9%).

CRYSTALLURIA, CYLINDRURIA, AND HEMATURIA HAVE BEEN REPORTED

Other changes occurring in less than 0.1% of courses were Elevation of serum gammaglutam elevation of serum amylase, reduction in blood glucose, elevated uric acid, decrease in hemog bleeding diathesis, increase in blood monocytes, and leukocytosis

OVERDOSAGE

Information on overdosage in humans is not available In the event of acute overdosage, the stom emptied by inducing vomiting or by gastric lavage The patient should be carefully observed and gi treatment Adequate hydration must be maintained. In the event of serious toxic reactions fro hemodialysis or peritoneal dialysis may aid in the removal of ciprofloxacin from the body, parti function is compromised.

DOSAGE AND ADMINISTRATION

The usual adult dosage for patients with urinary tract infections is 250 mg every 12 hours For complicated infections caused by organisms not highly susceptible, 500 mg may be administered

Respiratory tract infections, skin and skin structure infections, and bone and joint infections with 500 mg every 12 hours. For more severe or complicated infections, a dosage of 750 mg may be hours.

The recommended dosage for infectious diarrhea is 500 mg every 12 hours

In patients with renal impairment, some modification of dosage is recommended (SEE ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION).

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CONVENIENT *B.I.D.* DOSAGE

Recommended dosage schedule

Infection Site*	Severity of Infection	Dos
Respiratory Tract* Bone and Joint* Skin/Skin Structure*	Mild/Moderate	500 mg
	Severe/Complicated	750 mg
Urinary Tract*	Mild/Moderate	250 mg
	Severe/Complicated	500 mg
Infectious Diarrhea*	Mild/Moderate/Severe	500 mg

*** Due to susceptible strains of indicated pathogens. See indicated organisms in Brief S**

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ARMY RESERVE MEDICAL PROFILE NO.3

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“I joined the Army Reserve shortly after completing my responsibilities as Chief of Staff of Franklin Hospital in San Francisco. I was intrigued with the idea of trying something different, such as Army Medicine.

“I find that the challenges and rewards of serving as an Army Reserve physician complement my civilian practice. For a number of years, I’ve been teaching as a member of the Clinical Faculty at the University of California School of Medicine, and I thoroughly enjoy the many teaching opportunities available to me in the Reserve. It is a rewarding experience to be involved in the training of Army medical students, interns, and residents. I also enjoy interacting and exchanging information with full-time Army physicians and seeing a wide variety of interesting clinical cases.

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Dr. Tipton and residents examining post-operative patient in recovery room.

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About the cover:
Goose in flight. Graciously furnished by John Hyde, Alaska Department of Fish and Game, Juneau, Alaska.

The poor have many faces...

She lost her job last week—and her health insurance. That might not be so bad if her husband were still working. But she no longer has a husband—just three children.

She's one of the several million women who are heads of households and the sole support of their family. She's also now one of the many million unemployed whose health care insurance has run out and who haven't yet gotten another job with replacement coverage.

When it comes to health care, the poor have many faces. Hers is one of them. As a nation, we cannot turn our backs on those who want and need the health care to which they have a right. We must guarantee their access to high-quality, cost-effective treatment. Isn't that our responsibility?

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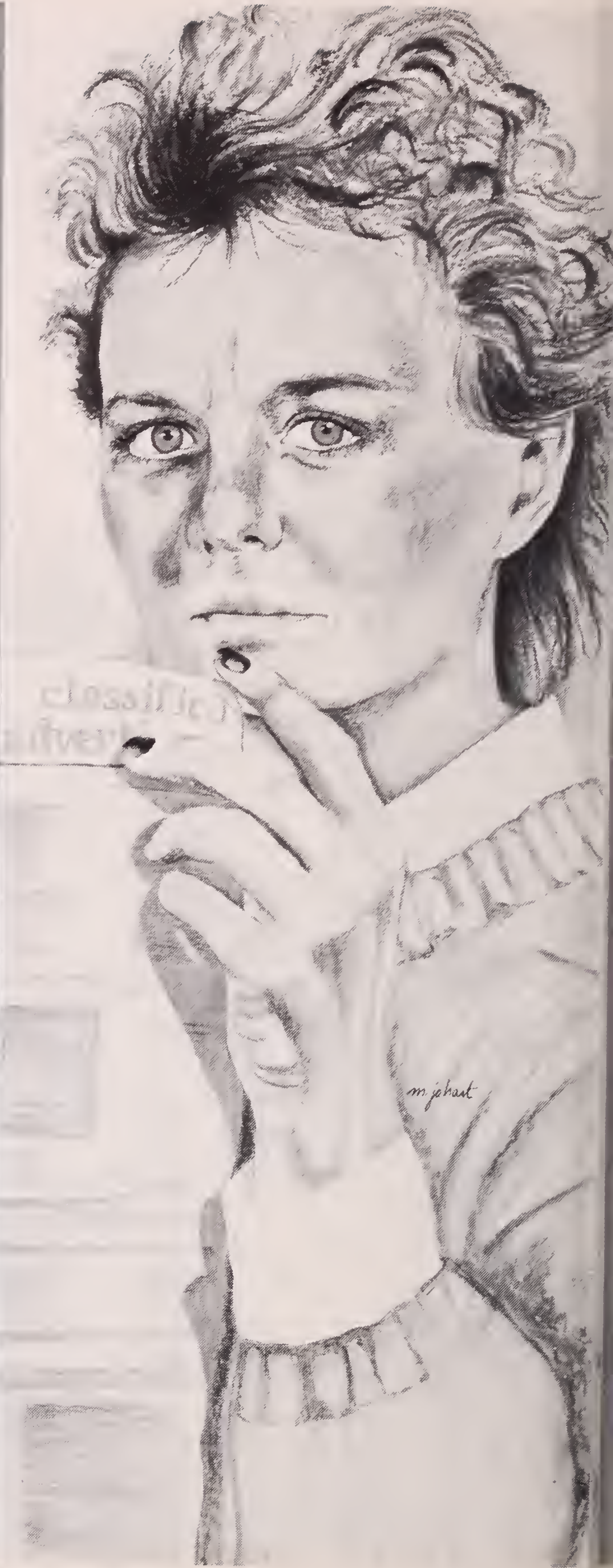

Cancer Causation Beliefs in an Alaskan Village

by Julie E. Sprott, R.N., M.A., Ph.D.[1]

ABSTRACT

At the request of an Alaskan village's traditional council that expressed a concern over a striking number of lung cancer diagnoses in the village in 1984–86 (n = 7 from a village population of 209), a case study was conducted to examine potential carcinogenic exposures of 13 persons who had been diagnosed with any cancer since 1972. In addition, 46 of 51 households were polled on current tobacco use and opinion on causes of the cancers.

The case study showed no consistent known carcinogenic exposures other than the smoking of tobacco for the lung cancer patients and the two others with cancers of the esophagus and bladder. Although the household survey indicated that at least one person in 74% of households believed that smoking causes cancer, just as frequently cited was their drinking water as a possible cause. The findings illustrate that strongly held rival hypotheses for cancer etiologies may coexist on a village level, and that uncovering these beliefs has important implications for planning cancer prevention programs.

PURPOSE OF THE INVESTIGATION

Since 1972, 13 persons were diagnosed with cancer in a predominately Native village in Alaska (population 209), seven of those with lung cancer within 1984–86 (Table 1). This prompted village-wide concern, such that the traditional village council made a request to investigate the matter to the Arctic Investigations Laboratory, Centers for Disease Control (CDC), Anchorage, and to an anthropologist working with the National Park Service in the region. Under the joint direction of the Arctic Investigations Laboratory's Acting Director and the field anthropologist, a nurse anthropologist (the author) was invited to conduct the investigation.

Table 1. Cancer Diagnoses in Villagers 1972–1986*

Year	Type of Cancer
1972	1 colon; 1 genital
1978	1 thyroid
1983	1 esophagus
1984	5 lung
1985	1 lung; 1 bladder
1986	1 lung; 1 stomach

*data provided by the CDC's Arctic Investigations Laboratory, Anchorage, Alaska

The purpose of the investigation was tri-fold: 1) to conduct a case study of cancer patients for residential, occupational, and recreational histories, including personal habits such as tobacco use in order to uncover any carcinogenic exposures, 2) to assess the current use of tobacco in the village, and 3) to cull the community's opinion as to possible causes of these cancers.

METHODS

Approval for the investigation was obtained through the traditional village council. Cancer patients or selected relatives were contacted, informed of the purpose of the investigation, and were asked to sign a consent before questions were asked of them. For the tobacco use and cancer causation opinion survey, villagers were approached household by household, and family members over age 18 were asked to sign a consent for themselves and for younger family members before being interviewed.

Standardized interview forms for the cancer case study were designed to tap smoking and chewing tobacco use, and to check for significant exposures to known carcinogens through residential, recreational, or occupational pursuits. Study of dietary habits was beyond the scope of this investigation. For the household surveys, family members were asked if they smoked tobacco or used smokeless tobacco. If so, they were asked to name types and brands, age began and duration of use, and intervals of non-use of three months or longer. The questions on cancer etiologies were designed to ascertain if villagers distinguished among causes of cancers, and also in particular, what they believed caused lung cancer. For both the cancer case study and the household survey, questions were presented verbally and the replies recorded immediately on paper. Cancer causation theories were noted with the household as the unit of response. A single household could be credited with a single response (one shared by all family members present), or with several different responses.

Five cancer patients were directly interviewed; close relatives provided information for those who were severely ill or deceased. All 46 households with family members present in the village at the time of the household survey were polled. Based on number of full-time residents (n = 209), information was obtained either directly or indirectly on 190 persons (91% sampled).

---

[1] Assistant Professor, University of Alaska Anchorage, College of Nursing and Health Sciences, Anchorage, Alaska.

Table 2. Smoking Habits of Residents Diagnosed with Lung Cancer

Code Number	Age Began Smoking	Number of Years of Smoking	Packs per Day	Pack Years
1	10	53	1	53
2	25	18	1/2-1	9-18
3	12	52	1/2	26
4	12	60	1	60
5	17	53	1	53
6	21	37	1 1/2-2	55.5-74
7	16	20	1 1/2-2	30-40

RESULTS

As expected, findings from the cancer case study indicated that all seven persons with lung cancer had significant exposure to cigarettes in their lives (Table 2). Pack-years of smoking ranged from an estimated 9-18 to 74 in this group. Also, two others with bladder and esophageal cancer had smoked for many years; risk of acquiring these cancers increases with smoking (1). Nothing in their life histories implicated any risk factor as consistently as did the smoking of cigarettes. Of note, three of the remaining four cancer patients with different types of cancers not known to be cigarette related had also smoked for most of their adult lives. Their histories revealed no differences in habit or lifestyle that might have accounted for their specific cancers.

From the household surveys, it was evident that many villagers understood that smoking causes cancer, either as a specific agent (29 households) or as a general cancer-causing agent (5 households) for 74% of households polled. However, an equally strongly held conviction was that cancers in the village had been caused by the drinking water (refer to Table 3 for a listing of the six most commonly cited beliefs). Other less frequently proposed causes included fall-out from nuclear testing, change from traditional diet to processed foods, indoor air pollution, and outdoor air pollution with ozone layer depletion. All in all, villagers purported as many as 15 different categorical causes of cancer in response to the survey questions.

Table 3. Six Native Theories of Cancer Causation in Rank Order by Percentage of Households in Agreement

1. Drinking Water Contamination/Chemicals 34/46 (74%)
1. Smoking Cigarettes 34/46 (74%)
2. Fall-out from Nuclear Testing in China or Russia 14/46 (30%)
3. Change from Traditional to "Western" Diet 8/46 (17%)
4. Indoor Air Pollution from Wood Smoke, etc. 7/46 (15%)
5. Outdoor Air Pollution and Ozone Layer Depletion 4/46 (9%)

DISCUSSION

The discussion section details the results of the cancer causation beliefs survey, because these findings present a unique village perspective that is rarely reported in the literature on this topic and is characterized by several noteworthy aspects. First, the list of the six most frequently mentioned cancer causation theories speak to a high level of sophistication and concern about cancer-causing agents by rural community residents. As do many in urban centers in Alaska, they too worry about indoor air pollution from wood stoves, outdoor air pollution, ozone layer depletion, and express concerns that a diet high in processed foods may increase risk of cancer. Additionally, they worry that exposure to wood-smoke during preparation of fish and game in the smoke-house might increase susceptibility to cancer. Fall-out radiation from nuclear testing in the 1960s was another concern voiced by villagers. They fear contamination of lichen and water with subsequent food chain pollution.

Not all of the Native theories of cancer causation can be adequately addressed in this limited space, but some seem less convincing than others. For example, while the impact to the lung of short-term, intensive wood-smoke exposure in smokehouses has not been studied in Alaska, excessive smokehouse exposure was not obviously singular to the lung-cancer patients in this village. I was informed that the majority of adult villagers engage in this traditional activity throughout their lives, and the case histories reflect this generalization. Likewise, radiation fallout is not a very likely etiological factor for cancer in this village—no such contamination has been recorded there in contrast to some other areas of Alaska. Even if it were a factor, one would expect to see a predominance of cancers of the breast, bone thyroid, and blood, rather than the single thyroid cancer detected in villagers so far (2).

Secondly, noteworthy are the quite disparate viewpoints voiced in the same household often by the same person: that village cancers were caused by smoking cigarettes AND by the drinking water. It was unexpected to find a community that generally ascribed to the scientifically accepted explanation that smoking causes lung and other cancers that also held the equally strong conviction that the drinking water was to blame. This situation is reminiscent of the phenomenon, "biculturalism," in which a culture bearer applies his or her traditional cultural values in one circumstance, then switches to seemingly conflicting or different values of another culture (usually of the dominant society) without experiencing the disparity.

On the one hand, it seemed that because tobacco could not be logically linked to all the cancers diagnosed in the village, another explanation was needed to fill the void of uncertainty. On the other hand, since many people in the village smoked cigarettes, it did not seem logical that some would acquire lung cancer and some would not—inconsistency was evident in light of the fact that 12 of the 13 persons diagnosed with cancer had smoked cigarettes for much of their adult lives, and yet only about half had acquired lung cancer. Additionally, some reasoned that cigarette smoking had been common in this village through several generations, yet not until recently had anyone been diagnosed with lung cancer. But given the remoteness of the village, it is doubtful that tobacco was as easily accessible 40 to 50 years ago as it had been in the last few decades with increased plane transportation to and from the village.

Reflecting the experience of the public in general, individuals who smoke or have acquaintances that continue to smoke without apparent ill effects are not easily convinced of the laws of probability as they pertain to lung cancer risk (3). A recent empirical study of the probabilities of premature death from smoking reported a rate of about 30% (4). This suggests that 70% of smokers will live out a normal life span, despite years of habitual tobacco use, and thus provide "examples" to smokers that may lull them into complacency.

From their standpoint, then, it is understandable why villagers would seek yet another ubiquitous environmental cause for the cancers as a means of coping with what seemed to them to be an ambiguous situation (5). The drinking water was mentioned as a possible cause by as many households as for smoking. But the meaning behind that reference was not entirely uniform. For 11 households, "bad water" was given as a response without elaboration; in 21 households, reference was made to the "old water system," when the village water pipe intake lay close to sewer pipe outflow and drainage from household dumping areas. In the 15-year span under the old system, villagers recounted many days of "bad tasting water," and "getting sick" periodically from the water—what appears to have been bacterial contamination of the water supply.

It is impossible to know if any type or degree of carcinogenic chemical contamination occurred at that time, although there is no scientific evidence to link such contamination with lung cancer (1). Nor is bacterial contamination of water thought to induce cancer (6). For three households, concern was raised that chemicals (fluoride, chlorine) currently added to water in the new system might have caused the cancers, another fear without basis (1,6). In sum, it appears that many villagers equated "getting sick" from the water in the past with potential to induce cancer over time.

The history of the village and its location generally weigh against the plausibility of water contamination as a cancer-inducing agent. The village is remote and without a history of proximity to massive chemical dumping by an industry. The geological formations preclude the presence of radon according to U.S.G.S. sources in Anchorage. Radiation fallout from prior nuclear testing in Russia and China and possible food chain and ground water contamination have not been documented in this region as mentioned earlier. Although it was not routine to test for certain carcinogens in the drinking water under the old water system, such tests recently performed on the current water system show no evidence of the presence of carcinogens.

It would be unfair to omit reference to changes in villagers' behavior regarding tobacco use, particularly since a majority affirm that smoking is harmful. Following the increase in diagnoses of lung cancer, some concerned villagers have banned smoking in their homes, and some displayed "no smoking" signs in their homes (14 of 46 households). Most with a no-smoking policy had a close relative who had cancer. Many villagers (n = 37) have quit smoking, but interestingly, the majority of quitters did so prior to 1984-86 cluster of lung cancer diagnoses. A majority of adults in the village still continue to smoke, although averaging about half as many cigarettes smoked per day as cited for U.S. adults overall (14 cigarettes versus 31 per day) (7). While it is known that knowledge of the potential hazards of smoking does not necessarily correlate with willingness to quit (8,9), one could postulate here that the fear of cancer may have been a motivation for villagers to reduce the number of cigarettes smoked.

CONCLUSION

There is nothing new in the methods used in this investigation: seeking the community's point of view about health concerns has been promoted as a part of a standard community assessment in public health for years (10). The study of beliefs and attitudes has been deemed important in understanding processes of change toward healthier habits in individuals, represented by the Health Belief Model (11) and for groups or communities as expressed in the current literature in terms of risk perception and assessment (12,13). It is well known that people worry about hazards to health much differently than do the experts (14). One would predict in this setting that little headway would be made in a cancer prevention program without addressing the concerns of the villagers about their drinking water. Doing so would hopefully remove a barrier to the acceptance on a personal level that cigarette smoking represents the primary risk for lung cancer, enlarging the list of reasons for quitting and increasing the likelihood of attaining that goal (15).

ACKNOWLEDGMENTS

Names of villagers and the field anthropologist who were instrumental in laying the groundwork for this investigation are regretfully omitted in the interest of village anonymity. Aid in instrument design, data analysis, and many suggestions in editing were provided by the staff at the Arctic Investigations Laboratory, especially by Dr. Anne Lanier, Acting Director.

The opinions expressed in this paper are those of the author and do not necessarily reflect the views of the Indian Health Service, and the Centers for Disease Control, Arctic Investigations Laboratory.

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Differentiation of Fatal Neonatal Enzyme Deficiency and Sudden Infant Death Syndrome (SIDS)

by George W. Brown, M.D., F.A.A.P.[1]
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INTRODUCTION

A two-day old term male presented with irreversible coma to the Valley Hospital Emergency Room. He died three days later at Children's Orthopedic Hospital and Medical Center in Seattle, Washington with a clinical and biochemical diagnosis of Ornithine Transcarbamylase Deficiency. Since then, another six and 1/3-hour old term male died at Valley Hospital from Sudden Infant Death Syndrome (SIDS). Extensive laboratory and autopsy investigation, including death scene investigation, revealed no cause of death. While Ornithine Transcarbamylase (OTC) deficiency is quite rare, review of Sudden Infant Death Syndrome (SIDS) literature suggests that SIDS is even more rare in infants under one month of age.

Recent publications have prompted new forensic emphasis for investigation of infant deaths and abuse (1–5). Bass *et. al.* have stressed death scene investigation of all SIDS (2). In X-linked dominant OTC deficiency, males are severely affected. Heterozygous females may have mild cerebral dysfunction or be completely asymptomatic (6–7). Heterozygote females with OTC deficiency are probably the most common of all the urea cycle disorders. Autopsy in OTC is usually unremarkable. The pathophysiology of SIDS remains unknown, although a multi- factional cause appears most plausible by available data (8–13).

Case Reports

Case 1. The Gravida 4, Para 3 mother of a 4281 gram (9 lb., 8 oz.) term male elected lay midwife assisted home delivery. Examination of the newborn at approximately 30–hours age in the pediatrician's office revealed a normal appearing newborn with no signs of distress and a weight of 3970 grams (8 lb., 12 oz.). Neonatal ophthalmic drops and intramuscular Vitamin K were given. Drowsiness and lethargy developed gradually from 36 to 48–hours age. Airway support and management for possible sepsis was begun in the local emergency room. After transport to Anchorage's Providence Hospital Neonatal Intensive Care Unit, the newborn's condition steadily deteriorated. A finding of high blood ammonia level of 1788 units (normal 64–107) prompted transfer to the newborn and metabolic unit in Seattle for suspected inborn error of metabolism. A single volume exchange transfusion was given enroute to Seattle. Examination on admission to Children's Hospital revealed a comatose, 3820–gram term male on respiratory support with left thorax central venous catheter, blood pressure of 68/50, and enlarged liver. Prior myoclonic seizure activity had ceased except for occasional stimulated (non-repetitive) jerking of the arms and legs. There was no evidence of dysmorphism. Laboratory data were normal except for serum sodium 151 mEq/L, serum chloride 116 mEq/L, and arterial ammonia level of 812 units. Despite aggressive peritoneal dialysis, the ammonia level rose to 1199 units. Urine orotic acid level was 100 times normal. Plasma and urine amino acid studies confirmed the diagnosis of OTC deficiency. The infant's parents, understanding the hopeless prognosis, requested withdrawal of supports, and the infant died at 5–1/2 days (134 hours) age.

Case 2. The 36–year old Gravida 5, Para 2, miscarriage 1, abortion 1 mother of a 4423 gram (9 lb., 12 oz.) term male delivered in Valley Hospital Obstetric unit Birthing Room in Palmer. Prenatal course with normal interval visits was uncomplicated. The mother was blood type O, RH negative, immune to rubella, and had a non-reactive RPR. A genetic amniocentesis done in early pregnancy revealed the fetus to be a 46–XY Karotype. She was delivered with the fetus en caul (intact amniotic sac). Rupture of the sac produced clear fluid. The oropharynx was cleared by bulb syringe and Apgar scores were 8/9. Delivery was two days after EDC by dates and eight days after EDC by early ultrasound. Dubovitz score was 40 weeks. Initial neonatal transition was normal. The infant was placed on the Ohio warmer at 1–1/4 hours age because temperature dropped to 36.4° C (97.5° F).Temperature rose promptly to normal, and he was wrapped in triple blankets, fitted with a head cap, and taken to his mother. He nursed well at two–hours age and fell asleep on his mother's abdomen. She also fell asleep. At three hours and 23 minutes age the post-partum nurse went to check the mother. The nurse found the mother fully asleep and the infant deeply cyanotic, flaccid, and with no pulse or respirations. Prolonged resuscitative efforts for over three hours were not successful. Complete autopsy diagnoses: 1. "Full term male with SIDS at approximately 4-hours age," 2. "Moderate pulmonary edema of unknown cause."

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DISCUSSION

Diagnosis of OTC deficiency was made by urine, serum and skin biopsy amino acid analysis through the metabolic defect laboratory in Seattle. This rare X-linked dominant disorder is reported as usually fatal in affected males (14). It is too rare to be given an accurate incidence. Symptoms begin within hours or a few days after oral ornithine intake. Prenatal obstetric and genetic counseling is recommended for affected families. Newborn males of possible maternal carriers should have ammonia and amino acid levels determined a few days after oral feedings are started (7). Explanation of often ineffective treatment should be made for the parents. Careful birth control for maternal carriers is an ideal medical consultation goal. Newborn females of maternal carriers may be tested, and all females of child-bearing age in affected families would ideally benefit from genetic counseling and oral protein loading before urine orotic acid measurement (7,14).

Review of the family history of the OTC affected neonate (Case 1) described above revealed that a maternal uncle died shortly after birth of unknown cause. A maternal male cousin was stillborn at six months gestation after his mother had delivered normal term male and female newborns who remain healthy. Another maternal aunt delivered normal term male and female children who remain healthy. This male, now just over one year of age, had normal blood and urine amino acid testing. Efforts to have urine orotic acid screening of this infant's four aunts have not yet been successful.

A frequently stated period of vulnerability for SIDS is from two to six months. There appears to be a gradual decline in the United States incidence, approaching only 2/1000 live births during the last decade. Some reports of unexplained neonatal deaths have suggested SIDS may occur in the first week or so of life (1). Caution is needed before accepting SIDS as a final diagnosis. Detailed forensic pathology investigation, including death scene investigation, is preferred. Recent such investigations have uncovered several other causes of death, rather than the at first suggested SIDS diagnosis (2,3,5). Unexplained neonatal deaths should prompt detailed genetic and metabolic investigations (6,7,10).

Review of the family history of the apparent SIDS victim (Case 2) at 6 and 1/3 hours revealed a 39-year old maternal aunt with Gravida 4, Para 1, miscarriage 1, abortion 1 with one stillborn. The living child is a healthy male. The stillborn was male with no discernible cause of death by autopsy. Both this maternal aunt and the six and 1/3 hours apparent SIDS neonate's mother underwent orotic acid urine screening after a protein loading meal with negative results.

The pathologic diagnosis of "SIDS at approximately four hours age" was surprising to the clinicians and remains most doubtful. Open discussion with the infant's family stressed metabolic causes that could not be diagnosed post-mortem. Absence of any signs of Transient Tachypnea of the Newborn and Asphyxia during the clinical course made those diagnoses unsupportable. Thus, the final diagnosis of "SIDS," with the accompanying diagnosis of "Moderate pulmonary edema of unknown cause," is in no way proven. Discussion with these parents regarding risk of SIDS recurrence and the more rare risk of an undiagnosed metabolic disorder allowed them to choose another pregnancy which produced a healthy term female.

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A Tribute to Dr. Werner Henle Collaborator on Alaska Medical Research

by Anne P. Lanier, M.D., M.P.H.[1]

The death of Dr. Werner Henle on July 6, 1987, was a loss to Alaska as well as the rest of the world. Dr. Henle was a prominent virologist who, with his wife, Gertrude, determined that Epstein Barr virus (EBV) was the cause of infectious mononucleosis. They also identified the first virus (EBV) associated with human cancers (Burkitt lymphoma and nasopharyngeal carcinoma).

Werner Henle was born August 27, 1910, in Dortmund, Germany, the son of a surgeon and grandson of Jacob Henle, a famous anatomist–histologist. He earned his undergraduate and medical degrees from the University of Heidelberg. He came to the United States in 1936, joining the University of Pennsylvania staff that year and the Children's Hospital staff three years later. He became director of the Virus Diagnostic Laboratory, and he and his wife devoted their careers to the identification of viruses in culture and sera.

In the 1970s, studies in Alaska documented that the incidence of nasopharyngeal carcinoma (NPC) among Alaska Natives was over 15 times that of U.S. whites (1–3). Werner Henle began collaborating with the Arctic Investigations Laboratory, Centers for Disease Control and Alaska Native Medical Center to determine if EBV was associated with NPC in Alaska as it was in other high incidence areas. Over 6,000 blood and tissue samples were sent to the Doctors Henle for EBV testing. This research has shown that EBV is associated with nasopharyngeal carcinoma (NPC) in Alaskan Natives, and that it is also associated with an unusual type of salivary gland tumor ("Eskimoma" or malignant lymphoepithelial lesion) which occurs at high rates in Eskimos (1,4). Work has also shown that persons with EBV associated malignancies have EBV antibody patterns that may be useful in screening, diagnosis, and prognosis (5,6).

Doctor Werner Henle never visited Alaska, but did much for Alaska and for research in Alaska. His enthusiasm, ideas, and generous collaboration will be missed.

Doctors Werner and Gertrude Henle wrote the following story of EBV for a special lecture given at the First International Symposium on Epstein–Barr Virus-associated Malignant Diseases held in Loutraki, Greece, 1985. With permission of Dr. Gertrude Henle and the original publisher, the article* is printed here.

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Special Lecture*

Epstein–Barr Virus: Past, Present and Future

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At an occasion like this, it may be permissible to discuss the past, the history of the Epstein–Barr virus, from a rather personal and perhaps slightly biased perspective. This may be less than exciting for those who were among the pioneers in this field and present at the creation, but those of you who joined the steadily growing crowd at later stages might not be aware of how much of the initial research depended on being at the right place at the right moment, on recognizing and grasping an opportunity as it arose, or on just good old plain luck.

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Our interest in human cancer viruses preceded the discovery of the Epstein–Barr virus by several years. We had started to work with polyoma virus, a newly discovered mouse tumor virus, to become familiar with essential techniques, among them assessment of the role of interference and interferon in the establishment and maintenance of persistent viral carrier cultures and the use of immunofluorescence for monitoring primary and persistent infections.

We thus were not unprepared for the telephone call we and many other virologists received in the late 1950s from Carl Baker, then Assistant Director of the National Cancer Institute to invite grant applications for support of a search for human cancer viruses. They were thought to exist in analogy to the animal tumor viruses which were extensively studied as potential models for their as yet unknown human counterparts. However, the experiences gained were rarely if ever, applied to human cancers.

No detailed research proposals were required because there were no leads, after all, on which to base them; merely a brief outline of the proposed general approach was requested and, of course, a not too heavily padded budget. As an additional inducement, 10 years of committed support were promised. Can you imagine 10 years without writing a competing grant application?

Carl Bakers's telephone call translated our vague plans into action, and we received a 10 year grant entitled "Interference phenomena in the detection of human cancer viruses." Accordingly, we cultured numerous specimens from childhood cancers, supplied by the surgeon–in–chief of the Children's Hospital of Philadelphia, C. Everett Koop, who is now the U.S. Surgeon–General, and we tested the out–growing cells for resistance to vesicular stomatitis virus as an indication of interference or interferon production, induced by an indigenous cancer virus that might be present. We have still to find a single cell culture of man or beast, fish or foul, reptile or insect that was normally resistant to this omnipotent virus. The cultured childhood tumor cells, unfortunately, proved no exception; they failed to resist infection by vesicular stomatitis virus.

When Chick Koop returned in 1963 from a conference in Africa, he told us about Burkitt's lymphoma and urged us to work on it because the epidemiology of this most frequent tumor of African children strongly suggested that it was caused by a virus. We immediately wrote to Denis Burkitt and other physicians on the African scene only to learn that everyone was already committed to other investigators. We apparently had missed the boat.

A year later, Tony Epstein and his colleagues at the Middlesex Hospital in London had established continuous cultures of Burkitt's lymphoma cells and found, on electron microscopic examination, herpes–like virus particles in a small proportion of the cultured cells. This discovery aroused few ripples of excitement because most virologists assumed that the virus could only be herpes simplex, cytomegalo or chicken pox virus; none of these, nor any animal herpes virus was as yet suspected of causing cancers. The virus indigenous to the Burkitt cells was therefore thought to be a harmless passenger of no particular interest.

As a last resort, Tony Epstein sent the Burkitt cell cultures for identification of the virus to Klaus Hummeler who recently had spent a sabbatical in his laboratory and had been in charge of our virus–diagnostic service at the Children's Hospital of Philadelphia which had just been dismantled, because the Pennsylvania Health Department withdrew its support for financial reasons. Klaus Hummeler came to our office, waving the bottles, to ask what should be done with them. Whereupon my wife promptly responded, "give them to me," and thereby came our chance to work on Burkitt's lymphoma.

The title of our grant was not a mere figment of our imagination. My spouse showed that the EB–1 and EB–2 Burkitt cultures were highly resistant to VSV, that the cells, following implantation, conferred resistance to monolayer cultures of human, but not rabbit or mouse cells, suggesting production of an interferon which indeed was found in the culture media. Another Burkitt cell line serves now as a potent source of one type of human interferon.

My spouse was unable, however, to transmit the indigenous herpes virus to routine cell cultures or to chick embryos, hamsters or mice of all seasons, using all possible routes of inoculation. This failure provided the first clue that the virus in the Burkitt tumor cultures was previously unknown but for proof, immunologic procedures were required. Only immunoflourescence appeared to fit the situation at hand but where to get sera from Burkitt patients which we believed, alas mistakenly, were needed.

Our chance came when we learned that a Nigerian Burkitt patient had been flown to the Clinical Center at the National Institutes of Health in Bethesda for plasmapheresis and attempts to sediment the expected C–type Burkitt's lymphoma virus from the plasma by high-speed centrifugation. It is truly amazing how much debris, how many "virus–like particles," can be sedimented from anybody's plasma, but C–type virus particles have not been among them. When we called Ray Bryan, then the Director of the National Cancer Institute, to request the supernates from the centrifuge runs, I distinctly heard "My God, they pour them down the drain" — together with all the antibodies we needed. Luckily, one more run was planned and its supernate, labeled P–91, yielded brilliant immunofluorescence in about 10% of the EB–1 and EB–2 cells. A serum from an American child with leukemia, chosen as control, failed to react. The resulting euphoria was of only short duration, however, because it was soon found out that many American sera as well as commercial human gamma globulin, also gave positive reactions.

We had to prove that the immunofluorescence was referable to the indigenous herpes virus. For one thing, the percentage of immunofluorescent cells and the percentages of virus-producing cells in given Burkitt cell lines, and soon other lymphoblast cultures, were closely similar. When we presented a lantern slide which listed a dozen cell lines in descending order of the percentage of immunofluorescent cells at a conference of the American Cancer Society at Rye, N.Y., in 1967, George Klein was the first to rise to the discussion to exclaim that the same order applied to the cell membrane immunofluorescence which he had been studying, indicating that the cell membrane antigen was virus-induced. This conference was the most fruitful in our career because it led to collaboration with George Klein and his associates which has continued to the present day and resulted by now in over 75 joint publications, with more to come we hope.

Soon thereafter, it was shown by Gary Pearson, while a postdoctorate fellow, first in George Klein's and subsequently our laboratory, that the membrane-reactive antibodies were, in part, identical with EBV neutralizing antibodies.

With Klaus Hummeler we showed by negative contrast electron microscopy that viral nucleocapsids, extracted from Burkitt cells, acquired a fringe of antibodies, resembling the head of Medusa, after exposure to immunoflourescence-positive, but not negative sera. The antigen so detected was therefore named later viral capsid antigen to differentiate it from the diffuse and restricted early antigens discovered by us, and the EBV-associated nuclear antigen (EBNA) discovered by Beverly Reedman and George Klein.

Irrefutable proof of the identity of the immunofluorescent and virus-producing cells was obtained when Harald zur Hausen, as a postdoctoral fellow in our laboratory, picked individual fluorescent cells for embedding, thin sectioning, and electron microscopic examination. All were loaded with virus particles, whereas non-fluorescent cells, similarly prepared, showed none.

Soon after his return to Germany, Harald zur Hausen showed that the Burkitt cells which did not produce virus, nevertheless contained EBV DNA, and that Burkitt's lymphomas and nasopharyngeal carcinomas harbored EBV DNA in amounts equivalent to multiple viral genomes per cell. These observations have been amply confirmed by others with ever more refined techniques. Furthermore, any cell harboring EBV DNA was shown by the Stockholm group to express the nuclear antigen EBNA.

To return to immunofluorescence, we made, of course, all along efforts to properly identify the virus with the aid of acute and convalescent sera from patients with primary herpes simplex, cytomegalo or vericella virus infections, as well as with hyperimmune sera to various animal herpes viruses. To compress several months work into one sentence, the virus indigenous to the Burkitt and other lymphomablastoid cell lines was antigenically unrelated to any known herpes virus. It was, indeed, new and we named it—provisionally we thought—EB virus after the EB-1 culture in which it was first observed in order to retire the awkward designations "herpes-like," "herpes-type" or even "leuco" virus. A year or two later, we would have baptized it infectious mononucleosis virus, since all herpes viruses are named after the principal disease they cause, but EBV was already too well entrenched to make a change.

Why was EBV production limited to only a small fraction of the cultured cells? Was the autogenous interferon production the limiting factor or was an enzyme in short supply that another herpes virus could contribute? Helper viruses had just become fashionable, but those we tested, that is herpes simplex, mumps and reo-3 viruses, provided no help. However, help came unexpectedly when a batch of completed culture medium was kept inadvertantly in the warm room for a week instead of the 48 hours we used in the prelaminar flow hood days to eliminate bacterially contaminated batches.

Frugal as we were, we used this batch of medium and were surprised by 5- to 10-fold increases in virus-producing cells. We traced this enhancement back to a loss of arginine by action of a fetal calf serum component. We still use arginine-free medium today to raise the number of virus-producing EB-3 cells. Subsequently, various anti-metabolites were shown by others to induce cycles of viral replication in latently infected cells by as yet obscure mechanisms.

The immunofluorescence test was soon applied to seroepidemiologic surveys which showed that antibodies to EBV were detectable anywhere in the world, even in such remote regions as the Amazon jungle, South Pacific atolls, Aleutian islands and I am sure, Siberia if anyone should care to go there to collect sera. Depending on the state of hygiene, the degree of crowding, and other socioeconomic conditions, nearly everybody was found sooner or later to acquire antibodies to the virus. However, all sera from African Burkitt patients, kindly supplied now by Eva Klein and others, had substantially greater concentrations of antibodies than sera from African control children so that EBV remained a candidate for the etiology of Burkitt's lymphoma.

The association of EBV with nasopharyngeal carcinoma and with infectious mononucleosis were both discovered by chance. When Lloyd Old and his associates needed, and received sera from African patients with tumors other than Burkitt's lymphoma as controls for double diffusion precipitation tests with extracts of virus-producing Burkitt cells, they included sera from several patients with nasopharyngeal carcinomas. To everyone's surprise, these sera reacted like Burkitt sera, producing up to 5 lines of precipitation. When we tested the same and additional sera from nasopharyngeal carcinoma patients, we found that they reacted like Burkitt sera also in the immunofluorescence test, yielding much higher antibody levels than sera from patients with other head or neck tumors.

Because EBV had turned out to be one of the most widespread human viruses, we suspected that it might induce a common illness as its primary clinical activity. In our search for this disease we were assisted beyond the call of duty by one of our young female technicians who was seronegative, developed infectious mononucleosis, and seroconverted in its course. This was not a laboratory infection as proclaimed by the Division of Biological Hazards of NIH. Our technician was a very pretty girl and thus was exposed to the kissing disease virus by the natural route.

Before her illness, our technician had often donated leukocytes for experiments carried out by Volker Diehl, then a postdoctoral fellow in our laboratory. Her cells had never grown by themselves in culture; they grew only when co-cultivated with lethally x-irradiated Burkitt cells from producer cultures. Leukocytes donated by her during her illness, however, now grew readily by themselves, yielding permanent EBV-positive cultures. These were the initial clues for the causal relationship of EBV to infectious mononucleosis and for the lymphoproliferative effects of the virus.

We then remembered that our friends at Yale, James Niederman and Robert McCollum, had collected sera from freshman at entry into college and again from those who developed infectious mononucleosis during the ensuing four years in anticipation of future candidate viruses for the etiology of the disease. They sent us coded sera and all pre-illness sampled were readily identified by us; they were negative in the immunofluorescence test, whereas all acute convalescent phase sera were strongly positive.

As mentioned, four distinct groups of EBV-associated antigens had been identified in time, and differentiation of the immunoglobulin class of the corresponding antibodies added another dimension to the EBV-specific serology. It was found that each of the three EBV-associated diseases (IM, BL, NPC) evokes at its height a characteristic spectrum of antibodies which differs from the other two and the pattern seen in healthy persons after long-past primary infections. IgM antibodies to the viral capsid antigens are limited to infectious mononucleosis; high IgG antibody titers to the viral capsid and restricted early antigen are characteristic of Burkitt's lymphoma; high titers of IgA and IgG antibodies to the viral capsid and the diffuse early antigen are an outstanding feature of nasopharyngeal carcinoma.

The EBV-specific serology thus became an important diagnostic tool. In the tumor patients, it provides, furthermore, information on their prognosis and serves to monitor them during remissions for evidence of imminent tumor relapses. Most recently surveys for IgA antibodies to viral capsid antigen and IgG antibodies to the diffuse early antigen have been used successfully in China and recently also Alaska for early detection of nasopharyngeal carcinoma patients.

All these studies depended on efficient, unstinting, harmonious collaborations on a world-wide basis with clinicians, epidemiologists and scientists covering many disciplines. It has been our pleasure and a rewarding experience to work closely with numerous individuals on all continents, some of whom we have yet to meet in person. Through the combined efforts, EBV has become a highly respected and fascinating virus which has attracted the attention of literally hundreds of molecular virologists, immunologists, biochemists, cytogeneticists and others.

The present, therefore, is as exciting as was the past. The current symposium bears witness to that and has left little for us to add except to ask, perhaps, a few questions.

We still need to know more about events during the incubation period of infectious mononucleosis. If the primary targets of EBV are pharyngeal epithelial cells, as seems likely now, why is the incubation period of infectious mononucleosis 4 to 7 weeks instead of the few days observed with the usual respiratory viruses?

We have learned that 13 days after exposure, or 25 days before onset of illness, EBV-carrying lymphocytes were already circulating in the blood of a most cooperative, susceptible Swedish boy, who in the course of a party had kissed a girl who two days later developed signs of infectious mononucleosis. We need to enlist other susceptible and cooperative contacts of cases, as hard as they are to find, and study them within the first few days after exposure, including a search for virus in the oropharyngeal secretions.

What is the actual incidence of chronic active infectious mononucleosis? Are all cases due to EBV or are some due to other viruses that have been aided by the immunosuppresive effect of a primary EBV infection?

How many of the polyclonal lymphoproliferations observed in immunologically compromised patients are EBV-induced? In observed in organ transplant patients, how often do the lymphomas arise from the recipient's cells and how often from the cells of the organ or blood donors?

Regarding Burkitt's lymphoma, we still do not know many details of its genesis. Is EBV one of several initiators of this malignancy or, in other words, are EBV-associated and non-associated tumors the same disease or two distinct entitles?

Are the characteristic chromosomal translocations in Burkitt cells truly mere chance events due to enhanced lymphoproliferation induced by malaria, or the acquired immunodeficiency syndrome, or any other excessive antigenic stimulation? Is the translocation of the c–myc oncogene from chromosome 8 to the immunoglobulin gene regions of chromosome 2, 14, or 22 the final step or does the oncogene product set off yet another step in the chain of events leading to the development of the tumor?

The course of events leading to nasopharyngeal carcinoma is even less clear. What is the cell or origin? Does the carcinoma arise from fetal thymic remnants in Waldeyer's ring, as has been suggested many years ago, and supported now by reports of an association of EBV with some thymic, salivary glands and possibly also some tonsil and larynx carcinomas? All the anatomical sites involved are derived embryologically from the same region, the third and fourth primitive pharyngeal pouches.

While certain epithelial cells have been productively infected with EBV in vitro, there is as yet no evidence of immortalization of epithelial cells by EBV. This should not be too surprising because nasopharyngeal carcinoma cells have not been grown permanently in culture. Once the appropriate growth factors for the carcinoma cells are identified, they presumably will support also the growth of experimentally EBV-transformed epithelial cells.

Despite the deficiencies in our knowledge, one wonders why a key role of EBV in the genesis of Burkitt's lymphoma and nasopharyngeal carcinoma is still questioned, whereas a causative role of the fourth "first human cancer virus," the human T–cell leukemia virus, tacitly is accepted as the cause of human adult T–cell leukemia, even though it should raise similar doubts. In fact, EBV has served as model for the HTLV studies; every seroepidemiologic observation made with HTLV is matched by earlier observations made with EBV. This includes the existence of many seropositive healthy viral carriers which clearly implies that in addition to HTLV other factors are needed to induce a T–cell leukemia or lymphoma.

Truly amazing strides have been made regarding the biochemistry and molecular biology of EBV. The viral genome has been deciphered, fragments of it have been cloned, genes have been transfected to various types of cells, new EBV–specific antigens have been identified, and a number of viral polypeptides and glycoproteins are produced now in quantity for study of their biological functions, their usefulness in the specific serodiagnosis and in monitoring of EBV–associated diseases, and last not least, for production of a vaccine.

We have no doubt that the glycoprotein vaccine now in the offering will protect susceptible college students, at least transiently, against infectious mononucleosis, perhaps even permanently, if a subsequent exposure to EBV evokes a rapid recall of antibodies to intercept disease, although not necessarily inapparent infection. If thus a persistent viral carrier state ensues it will convert an initially transient into an ultimately permanent immunity. However, for prevention of EBV–associated malignant diseases presumably also latent persistent EBV infections have to be prevented, which presents a vastly more difficult task.

It is clear that one cannot discuss the present without at least a few glimpses into the future. We have learned in the late 1950s, however, that it is hazardous to go further. At that time, Sir MacFarlane Burnet, the eminent Australian virologist–immunologist and Nobel laureate, passed through Philadelphia and told us that he was abandoning virology to return to his first love, immunology, because all important things that can be done with viruses had been done. It showed that the crystal ball of even the great can at times be awfully cloudy. One thing we can say today with assurance; not all important things that can be done with EBV have been done. There is plenty of work ahead so that EBV will remain an efficient travel agent for all of you for some time to come. EBV has taken many of us all over the world, to study EBV–associated diseases in endemic regions of Africa or the Far East, or to attend EBV–devoted conferences, symposia or workshops in many parts of the world, including now this lovely spot in Greece at the shores of the wine blue sea. There are still further years of further EBV–induced travels ahead before the glycoprotein vaccine will put an end to it.

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Electronic Information for Physicians: A New Dimension in Solving Traditional Problems

Part II

by William A. Yasnoff, M.D., Ph.D.[1]

Last time (Ak Med 1988; 30(4): 135–137), the capabilities of AMA/NET to assist with diagnostic and therapeutic problems were discussed.

III. Current Awareness of Specific Medical Topics

Physicians often need to obtain the latest information in a particular medical subject area. This may be a particular disease state, subspecialty, or a topic of current interest. Traditional sources for such information are the many available medical news publications, scientific meetings, and current medical literature. However, it is often very difficult to sort through the plethora of available information to find the specific items of interest.

This is another area where electronic information can be helpful. AMA/NET provides a number of services which directly address the need for current medical information. One of the most useful of these is the Associated Press Medical News Service, which provides up to the minute reports of medical news as they are transmitted on the Associated Press newswire. These stories are updated continuously, and are available on both a chronological basis and by subject area. Medical news stories are thus available almost immediately on AMA/NET.

Another useful source of current information is the literature update by specialty. This provides, via a search of EMPIRES, the most recent literature in a given specialty area. The social and economic aspects of medicine (SEAM) database, which is maintained by the AMA, is another useful source of information. This database references selected articles in both medical and nonmedical literature which pertain to social, ethical, and legal issues related to the practice of medicine. Topics of current controversy are often represented in this database.

An additional source of current information in AMA/NET is the public health information services. The Centers for Disease Control, the Surgeon General's office and the FDA all provide current information on AMA/NET. For example, the full text of the weekly Morbidity and Mortality report from the Centers for Disease Control is available.

These sources of information, combined with the literature searching capabilities, make it easy to obtain the latest information on any particular topic of medical interest.

IV. Reference Information

Reference information needed by physicians is traditionally available through a nearby medical library. An example of this type of information on AMA/NET is the CPT procedure codes. The CPT codes are used for reporting most medical procedures to third party payers. This can be useful when such a code is needed and the printed reference is not available.

V. Continuing Medical Education

Continuing medical education has become increasingly important as the rapid pace of medical discovery leads to proliferation of new knowledge and changes in previous concepts. The traditional methods for obtaining continuing education involve attending meetings, either locally or by traveling to a remote location, or reading material followed by subsequent self-administered examinations. More recently CME has been available in videotape form.

Electronic information has a role to play in CME, also. In particular, computers are well suited for patient simulation type of exercises where the effects of many different patient management scenarios can be explored safely and easily. AMA/NET provides access to a number of such simulations which have been developed at the Massachusetts General Hospital. Examples of these include abdominal pain, diabetic ketoacidosis, and hypertensive emergencies. With these programs, access to electronic information can provide new opportunities in continuing medical education.

VI. Communications with Colleagues

Traditionally, physicians' communication with their colleagues has consisted of scheduled meetings, either local or remote, chance meetings in the hallway ("curbside consults"), telephone communication (which is very inefficient), and communication by letter, which is frequently used to report the results of patient referrals.

AMA/NET provides access to a new form of electronic communication: electronic mail. With electronic mail, messages are sent immediately, but stored so that each person sends and receives information at their convenience. This greatly simplifies written communication with colleagues since sender and receiver do not need to be simultaneously available. Thus, electronic mail can provide the speed of the telephone without the associated inconvenience and delay, and represents a cure for chronic telephone tag.

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[1] American Medical Association

Electronic communication can also take the form of an electronic bulletin board, allowing announcements to be made which are immediately accessible to system users.

VII. Non-Medical Information

Physicians also need information that is not medically related. Obviously, there are a multitude of traditional sources for all types of information via newspapers, magazines, other periodicals, and television. Electronic information provides a supplement to these sources. In particular, AMA/NET provides on-line access to the Official Airline Guide, which is very useful in planning travel, as well as various types of financial information. The latter includes business press releases, financial and commodity news, tax notes, and stock and commodity quotations. It is also possible to obtain information about investments managed by AMA Advisers.

VIII. Conclusions

Electronic access to information provides an important adjunct to traditional methods. In some cases electronic information provides capabilities which are not otherwise available. In the future, new forms of electronic information will be available. One such form is the "interactive reference," which provides referenced information tailored to an individual patient's situation. With such a reference, the process of extracting information relative to a specific patient from a number of sources is performed automatically providing both convenience and time savings.

This report has described some of the information sources and capabilities available on AMA/NET. It is clear that physician access to electronic information greatly expands, but does not replace, traditional information access methods.

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
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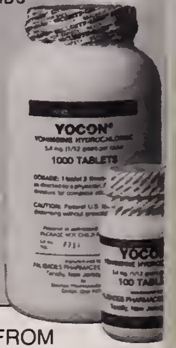


Program Notes
Arctic Investigations Laboratory
Centers for Disease Control

Prevalence of Antibody to Hepatitis A Among Alaskan Natives

Hepatitis A infection has a worldwide distribution, both sporadic and endemic, with a tendency for cyclic recurrences. Historically, this cyclic pattern has been evident in Alaska. Most recently, epidemics of disease were seen during the early 1960s and again during the mid 1970s, though not all of Alaska was hit by these epidemics. During interepidemic periods, isolated outbreaks were occasionally seen, and low levels of disease persisted in communities such as Anchorage.

Since the time of the last statewide epidemic, the pool of susceptible persons has been growing. Isolated outbreaks such as occurred in Manokotak in the Bristol Bay area during 1983–84 have occurred, but they have not spread. During the fall of 1986, the State Epidemiologist's office began to notice a dramatic increase in the number of Hepatitis A cases reported.

In order to learn more about the size and makeup of the susceptible population, the Arctic Investigations Laboratory (AIL), the Alaska Area Native Health Service, and the state Section of Epidemiology decided to test a sample of banked sera for antibody to the Hepatitis A virus (anti–HAV). The majority of the sera were collected as a part of the statewide Hepatitis B control program, begun in 1983, and are stored at the Arctic Investigations Laboratory.

An age– and community–stratified sample was selected. In general a 5% sample was selected, with a minimum sample size of 20 persons per village. The samples were equally divided into four age groups—persons born before 1948, born 1948 through 1962, born 1963 through 1972, and born 1973 through 1980.

Of the 178 communities with a 1980 Native population of at least 50, sufficient sera for the sample was available for 147 communities, representing 90% of the Native population. Four thousand–thirty sera were tested, with an overall positivity rate of 49.3% (Table 1). This rate varied significantly among the different service units from a low of 35% in the Anchorage service unit and the Pribilof Islands, to nearly double that in the Yukon–Kuskokwim Delta Region.

Table 1. Results of Anti–HAV testing

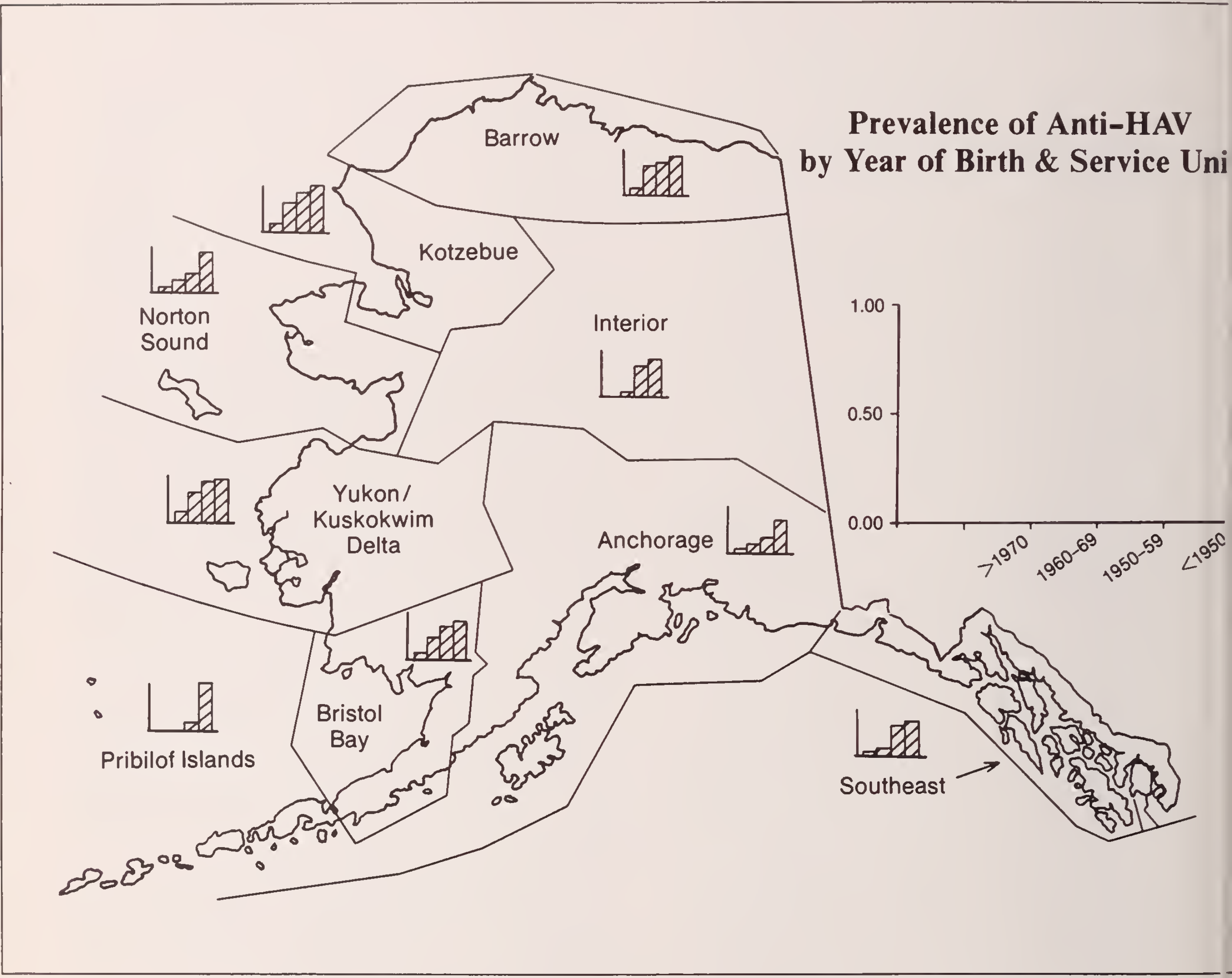
Service Unit	No. Communities Sampled	No. Sera Tested	% Anti–HAV Positive
Yukon–Kuskokwim	44	1,030	65.7
Kotzebue	12	308	64.6
Barrow	6	188	57.4
Bristol Bay	17	372	53.2
Norton Sound	14	348	44.3
Southeast	10	396	38.9
Interior	14	344	38.4
Anchorage	28	1,004	35.0
Pribilof	2	40	35.0
	147	4,030	49.3

The prevalence of anti–HAV increased with age. Statewide, it is a generally smooth rise from 6.3% among persons born since 1976, through 47% among those born 1961–1965, and reaching 86% among those born in 1945 or before. The precise pattern of increases varies among the service units (Figure 1), though increases with age are always present. (The service units along the northern and western coasts were hit by the epidemic in the mid–1970s and accordingly they have the highest positivity rates, particularly among younger persons.) At the level of the individual village, a precise estimate of the timing of the most recent epidemic can often be made. In 27 (21%) of the 129 communities for which the minimum sample of size 20 was drawn, there is no age overlap between anti–HAV positive (older) and negative (under) persons. Since the disease is highly infectious, the epidemic may have hit the village just before this younger group was born. In an additional 77 (60%) of these communities at most two of the 20 tested sera overlap in age with those of the opposite group. These may represent loss of antibody, absence from the village at the time of the epidemic, or persons who were not infected.

The proportion of positives within a given service unit is generally similar for most of the villages, but there are exceptions. One village in the Anchorage Service Unit was found to have no positives among the 20 sera tested. In Norton Sound (overall prevalence 44.3%) villages ranged from 25% to 95% positive. Most villages in the Yukon–Kuskokwim Delta Service Unit had high prevalence of anti–HAV and evidence of the epidemic during the 1970s; however, a group of upriver Yukon villages showed no evidence of infection since the 1950s.

The results of this testing emphasize the potential public health problem which hepatitis A infection poses for the Alaska Native people. Extrapolating the age-specific results of our serosurvey, we would estimate that 45% of the 1980 Alaska Native population were then susceptible to hepatitis A, some 29,000 persons. Since that time, approximately another 15,000 Native children have been born, all seronegative for hepatitis A. The virus is known to be highly infectious, and it appears that in the past the vast majority of susceptible persons in a village hit by an epidemic become infected. Many infections are asymptomatic, but overall morbidity is still high, particularly among adults who develop symptomatic disease.

Surveillance continues to monitor the possible growth of an outbreak. The Section of Epidemiology, Division of Public Health, State of Alaska, has published recommendations for control of the outbreak (see Epidemiology Bulletin, Vol. 1, week ending January 15, 1988). Please contact Lisa Bulkow (271-4011) or Sue Anne Jenkerson (561-4406) if you have any questions or comments regarding the HAV outbreak.



Prevalence of Anti-HAV by Year of Birth & Service Uni

History of Medicine in Alaska

JOSEPH RUDE, M.D.

During the summer of 1987, Kathy Hedges, then 19, interviewed Joseph Rude, M.D., who was then in his 90s. It was not easy to extract anything about Dr. Rude from him, but Kathy succeeded in making a delightful portrait. It is an account of a modest, knowledgeable, and exceedingly useful elder by a perceptive and appreciative young adult, much like the esteem an Alaskan Native holds for his elders.

Kathy, the daughter of Dr. Gary and Marge Hedges, was graduated from Juneau High School. She briefly attended Niagara University in New York State. However, she found the East too different to be comfortable and transferred to the University of Puget Sound. She has spent the last year abroad, at the Universities of Copenhagen and London.

Born in Norman County, Minnesota, Dr. Joseph Rude had no idea he would be one of the few doctors to come to Alaska and stay.

Dr. Rude was born into a family with a strong Norwegian background. In 1872, his dad arrived in Redwing, Minnesota by steamer, and in 1897 his mom sailed up the St. Lawrence River.

Dr. Rude is a very friendly gentleman. When talking to him for this interview, it was very hard to get him to talk about himself. A modest man who happily shares what he knows and who is proud to tell you about his two sons and two daughters or the involvements of his wife, he is reluctant to speak of his own experiences. Dr. Rude grew up in a strong family and brought this strength into his own family. Maybe this is what helped keep him in Alaska for so long when it was a Territory, a land thought by many to be too harsh to live in.

Without completing high school Dr. Rude joined the Army during World War I and worked in the Army hospitals. There as a medic, known then as a "pill pusher," Dr. Rude spent his time taking care of wounded soldiers from France. It was here that Dr. Rude began his liking for medicine—for the chance to help others. As he remembers it, there were "...no nurses, men had to do pretty much everything."

Since he had not finished high school, Dr. Rude needed some courses before he could attend college. For this, he went to the Lutheran Bible School and Academy in Grand Forks, North Dakota, attending the academy half time and working as manager of a club to pay for his room and board. Two years later, he received his diploma.

With the completion of his academy years, Dr. Rude headed to the University of Minnesota. He started college by marrying his wife, Amy. They have been married for 67 years. After completing three years of pre-med, Dr. Rude joined 500 applicants for the 120 positions open at the University of Minnesota Medical School. He made it!

When Dr. Rude completed his four years of medical school, he took a one year internship at Fairview Hospital in Minneapolis. His training was in general medicine. Dr. Rude remembers his internship as being good training. During the 12 months of his internship, he delivered approximately 100 babies.

At the end of his internship a chance came to go to Ketchikan for $250 per month. The year was 1929, and he headed for "Lady Arctic." At this time there was no distinction between the Arctic and Southeast Alaska.

He moved on to Petersburg to go into practice on his own. It was good at the start, but soon the Depression hit. To keep his family going, Dr. Rude gladly took beef, venison, lamb or anything from farms for pay. He remembers that patients at Pt. Agassiz were always happy to pay in produce.

The Rude family spent 12 years in Petersburg. At that time, Dr. Doss of Juneau, the only American College of Surgeons member in the Territory, was thinking of retiring and wanted someone to take over his practice. So, Dr. Rude picked up his family and moved to Juneau where he has been ever since.

When Dr. Rude moved to Juneau in 1941, he worked at the Juneau Doctors' Clinic with three or four other physicians. He remembers long hours and Sunday afternoons at the Clinic.

Not all was work in Dr. Rude's day. In Petersburg, he owned a speed boat. He was often in races in either Petersburg or Wrangell. When he moved to Juneau, he sold the race boat and got a 28-foot gasoline launch, the "do-boy." With the "do-boy," Dr. Rude occasionally took trips to Angoon and Hoonah. He remembers making 100 trips to Skagway before there was a doctor there.

In 1964, Dr. Rude began to retire. For months he examined young people in Bolivia at colleges. There were more than 1,000 students to examine, plus clinics to hold in small churches on the outskirts of Cochabama for people who had never seen a doctor.

Even today Dr. Rude is keeping himself busy medically. Each year he is involved in giving physicals to young athletes in the Juneau school district. Even with all of this work, Dr. Rude still considers himself retired, because he has no office.

What Dr. Rude does have are his awards to show for all his work. Hanging in his living room are many plaques. One states that Dr. Rude was the "Juneau Man of the Year, 1965." In 1966, he was given the Christian Citizen Award from Sheldon Jackson Junior College, Sitka. In 1970 he gained the A.H. Robins Award for Community Service from the Alaska State Medical Association. But above all these, the award Dr. Rude is especially proud of is his Honorary Doctor of Humanities, received from the University of Alaska in 1985.

In addition, Dr. Rude stays active as a member of the Elks and the Boy Scouts of America. When the snow is good, he will grab his skis and head for the hills for some downhill skiing. In summer Dr. Rude will make rounds in his garden to see how his trees are doing.

What keeps Dr. Rude in the practice of medicine after all these years is his desire to continue to help others. He just enjoys taking care of his patients. He especially enjoys making house calls. As he says, "You really get to know the family, a means of making good friends." One of the most pleasant aspects of his medical career has been the ability to bring so many babies into the world.

In 1987, Dr. Rude joined 400-500 other Rude family members for the fourth family reunion in Beresford, South Dakota. In 1989, he plans to go again to a town near Fargo, North Dakota. Dr. Rude attributes his 93 years by careful living, no drinking, no carousing, and no cigarettes.

If his past is any indication of his future, we should expect to see Dr. Rude up on the ski slopes again next winter.

Kathy Hedges

Dr. Joseph Rude recently recounted the best story from his practice.

While in Petersburg, he had a patient who came from Germany. He wanted his hemorrhoids cured by an old German method consisting of inserting a bottle into the rectum and squashing the hemorrhoids against the bottle. Dr. Rude accommodated him but the bottle slipped inside and became stuck. Dr. Rude put him in the hospital and delivered the bottle through the abdomen under general anesthesia. The patient survived to tell the tale in the local bar over his beer. Dr. Rude submitted no bill, but later whenever he visited Petersburg the German insisted on giving him $5 for each year elapsed since Dr. Rude's last visit. Once after a four-year interval, he paid $20 on the nonexistent bill. Dr. Rude gave this to a church, but he is still uneasy about it as the patient was an atheist.

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Federal and many state legislators are putting physician office laboratory regulations into effect. The College of American Pathologists' EXCEL program currently meets most physician office laboratory regulatory requirements and is a convenient and economical way to prepare for expanded governmental monitoring.

For ordering information, contact the CAP Surveys Order Department, College of American Pathologists, 5202 Old Orchard Road, Skokie, IL 60077, 312-966-5700.

Glimpses of Alaskan Medical History

Edited by Robert Fortuine, M.D.

Hard Times at Unalaska (1792)

In August 1791 Captain Joseph Billings sent his ship *Glory of Russia* from the Chukchi Peninsula to Unalaska under the command of Lt. Gavriil Sarychev. Also on board was Martin Sauer, an articulate Englishman who served as secretary to the main expedition. Both their ship and another Russian vessel spent the winter of 1791–92 in Iliuliuk Bay near the present-day city of Unalaska.

Sauer's account of the voyage, which is excerpted below, tells how he and a few others lived ashore during the winter and adopted many of the features of an Aleut diet, including the use of raw fish, mussels, and limpets, and the use "instead of tea in the morning, [of] a teaspoon of essence of spruce in a small tea-kettle full of boiling water." This latter remedy could not have been Aleut, because spruce trees did not grow on the island at that time. He also collected "wild cresses" on a daily basis for use in a salad. These modifications of standard shipboard fare largely protected Sauer and his immediate circle from the worst effects of scurvy, which overall claimed the lives of 17 men before the ships set sail for home in mid-May.

Sauer was one of the few 18th century visitors to Alaska to give full weight to the role of diet in the prevention and treatment of scurvy. Most others attributed the disease to the damp climate, poor ventilation, and lack of exercise.

"The year 1792 had commenced with the most inauspicious prospects that the imagination can conceive: except myself, I believe, every one of our Company was affected more or less with the scurvy; some without any other outward marks than a sallow complexion, accompanied with shortness of breath, and an extreme lassitude of the whole frame, which prevented their taking ordinary exercise, or even walking far; some had small livid pimples all over the body, particularly about the legs, with soreness and violent itching; some had large livid blotches on their legs, arms, and other parts of the body; some were bloated all over, and almost all had their gums swollen to such a degree, that they nearly hid their teeth. Thus situated, it was with difficulty that we could muster able hands enough to hoist a cask of water on board. . . .

"The prospect before us grew more melancholy as the season for our departure advanced. More than three-fourths of our Company were confined to their hammocks by the scurvy; but our Surgeon, Allegretti, was among the number of those who, with the assistance of crutches, were enabled to move from place to place; and he, with Mr. Bakulin, was taken into our hut. Notwithstanding every possible method was adopted by our surgeons to check this disorder, it raged with unabated violence; and, toward the latter end of the month of February, we sometimes buried three men in one day; and the most athletic in appearance were the earliest victims. It was equally destructive to those who dwelt on shore, as to those whose birth was on board ship. At this time we began to doubt the possibility of ever leaving the island. I still continued my walks, but found some difficulty in rambling over the mountains; it certainly fatigued me more than usual; but I did not on that account shorten my excursions.

"Early in the month of March, the wind, which had hitherto blown from the northern quarters, veered to the southward; although rainy and misty weather continued during the day, the fogs were less and the nights more clear. We now observed, to our inexpressible joy, that the mortality ceased; that those who were violently afflicted with the scurvy did not get worse, and that no more were laid up; and soon after, appearances indicated returning health. The inhabitants of the island, with the natives who brought us their tribute, also supplied us with abundance of halibut and cod; the wild mustard growing about the habitations was gathered, and distributed to the different messes; and we again revived, in hopes of better days than this island could afford. . . .

"During the month of March the scurvy seemed perfectly at a stand, neither increasing nor diminishing materially upon the afflicted; but in early April, when the new plants produced a supply of vegetables, those sick who had used crutches were enabled to relinquish them, and willingly gave them up to such as began to creep out of their hammocks."

REFERENCE

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President's Page

Today is an uneasy time for doctors practicing medicine. Malpractice attorneys stay busy. Recently several of the tort reform laws passed by a few states have been found unconstitutional. Our rapidly advancing technology increases our ability to treat satisfactorily, but carries with it a host of complex ethical problems which we are far from solving as yet. As if we did not have enough problems, Mr. Dukakis recently declared that, if elected president, he will propose a national health care system just like the one in Massachusetts. However, before applying to law school or simply going fishing it may help to look backward to see if we can gain a little perspective on the current state of our profession.

I read history, but not because we have been warned that those who cannot remember the past are doomed to repeat it. I have seen very little that persuades me to accept the proposition that knowledge of the past protects us against repeating the errors of the past. I, at least, seem to go along making some of the same mistakes over and over, each time assuring myself that next time will be different, of course. The awareness of what has gone before has no prophylactic value as far as I can tell. Nonetheless, I find the reading of history reassuring.

To me there is comfort in knowing that however bad our current situation may be concerning our profession, the state of the nation, or our 12-year old's most recent report card, we can easily find a time within the past century or two when things were worse. Somehow the earth, human race and medical profession survive. This is not a realization which provokes ecstasy, but it does help to keep down hysteria.

Consider the problems of the practicing physician in the 19th century. The primitive state of medical knowledge at that time, compared to our own as we near the end of the 20th century, comes to mind immediately. Social and political issues, different from than those today, but perhaps as troubling, complicated the doctor's life then as now. Narcotics were uncontrolled and opiate addiction by the mid-19th century was known as the "American disease." Licensing laws did little to separate the qualified physician from the quack or the simply incompetent medical doctor. Additionally, the 19th century was the heyday of the pseudosciences; homeopathy, mesmerism, phrenology, spiritualism and the water cure all competed—vigorously—with medicine.

At one point in the later part of the 19th century homeopathy made a strong bid to become the dominant force in American medicine. However, today these practitioners are gone. But, in the course of dealing with these other disciplines, the mainstream of medicine had to examine their propositions, refute what was obviously not true, and accept and work with anything which they offered that seemed to help in the practice of medicine.

In this process, organized medicine gained a great deal. Cerebral localization of function evolved from the absurdities of phrenology; the importance of the unconscious in mental functioning became apparent when studying patients in mesmerizing trances. The abuse of illicit drugs, although still a major problem, is, believe it or not, less severe a problem than it was a hundred years ago.

It would be too much to hope that the bureaucrat and malpractice attorney would go the way of the phrenologist. Probably it would not be good if they did. For all the hassle they cause us, they obviously point in the general direction of some major problems that modern medicine has. Certainly medicine will prevail; I have no fear that the bureaucrats and attorneys will completely submerge us. Hopefully, in the process of learning to accommodate ourselves to the new realities of medical practice, we will learn something useful to us and to our patients. Perhaps we will be reminded of the importance of the art of medicine. Medical science has made such tremendous gains in the past century that it is easy to forget that science is only a portion of the knowledge necessary in order to practice medicine at the high level we all aspire to.

Irvin A. Rothrock, M.D.
President, Alaska State Medical Association

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Executive Privilege

I recently had the opportunity of reviewing a speech delivered by Dr. John Kitzhaber, an Oregon physician and current president of the Oregon State Senate. It offers a most clear and cogent presentation on the issue of uncompensated care. I would like to summarize his comments and add my thoughts. I hope you will take the time to read them thoroughly. It speaks to an issue that we in the provider community are just now beginning to understand, and it points out the need for greater dialogue between provider and legislator in order to address a critical social problem.

Let me begin my comments by stating to you that unequivocally the most serious threat facing the medical profession today is uncompensated care. If left unresolved it will erode the health of our society and, in turn, will lead to an erosion of the clinical autonomy of physicians and other providers. It will also undermine some of the very principles on which our health care system has been built and will lead to increased regulation of the practice of medicine and probably to a government controlled health care delivery system.

To understand the threat and challenge it poses, we must first consider the evolution of our American health care system. Our health care system was founded on the principle of universal access; the idea that ill Americans, regardless of their income, should have access to the health care system and to all the services it offers. We were able to deliver on this social objective because of our fee–for–service reimbursement system and the ability to cost shift. So when the poor came for treatment the service was rendered, and the cost was merely shifted to someone who could pay through an incremental increase in their bill or in their insurance premium.

This policy was no accident, but was the result of conscious decisions in both the public sector and the private sector. In the public sector, we enacted Medicare and Medicaid in 1986 extending coverage to the poor and the elderly. At the same time, we had a rapid expansion of private policies funded primarily through employment. This rapid growth of third–party insurance coverage led to the belief that health care for the poor in this country was free, when in fact it was being subsidized primarily by the government and by the business community.

We created what we felt to be an ideal health care system. It was a system with no financial restraints where individuals had access to as much health care as they wanted or needed. Physicians could practice pure medicine, viewing their patients primarily from their health needs without concerning themselves over income.

But this system also led to and encouraged utilization. It led to the deeply held social belief in this country that health care is a right. That resulted, understandably, in a dramatic increase in expenditures. The amount we spent on health care grew from $75 billion in 1980 to nearly $500 billion today. And, more telling is the growth of health care expenditures as a percent of the Gross National Product. We spent 7.4 cents on the dollar in 1970, and we spend about 11 to 12 cents today. If this rate continues by the turn of the century we will be spending 20 percent of the Gross National Product on health care, and by about 2020 we will be spending 40 cents out of every dollar on health care. This, of course, will not happen. It makes a great deal of sense in terms of social policy, but makes little sense in terms of an economic policy. No single set of expenditures can grow at a rate faster than the growth of the Gross National Product.

A number of factors have occurred that bring our ideal health care system into a collision with economic realities. New medical technologies, an aging population, and the federal budget deficit were some of the realities that were driving the costs higher and higher. The business community now became interested in cost containment in order to remain competitive worldwide, and government became interested in balancing the budget. The object of business and government was simply to reduce the exposure to the cost shift, and to reduce their funding and subsidy of the cost to care for the poor. The subsidy was not taken out of the system, it was merely shifted on to individuals and providers. How did they do it?

In 1983, the federal government enacted the DRGs which is a prospective reimbursement system that shifted economic risk on to providers. The private sector reacted exactly the same way with an increased involvement in HMOs, PPOs and other prospective managed care plans that put the providers at risk. But, the important thing to remember is that cost containment reflected absolutely no social policy beyond cutting costs for the government and the business community.

Our ability to deliver on the concept of universal access has depended on cost shifting and the willingness of business and government to subsidize the cost of care for the poor. But what we are seeing today is a progressive shifting of the responsibility to pick up that cost. Remember, that prior to 1980, the subsidy was borne by government and business which spread it out over taxpayers in general. So society was paying for the social responsibility to have universal access in this country. Why should we be so concerned about the shifting of the responsibility to pay for the care of the poor? Because severe social and professional consequences will result.

The first social consequence is a result of the competitive, market-driven system in the provider community. Since market systems were not designed to foster social responsibility, it shouldn't be surprising that no one is competing for the poor. This leads to a second consequence which is a very real and measurable deterioration of health for a growing number of Americans. We have 40,000 neonatal deaths each year from the complications of low birth weight. Two-thirds of those mothers do not receive adequate prenatal care. Forty percent of the poor in America are children, and only one-third of them are covered by Medicaid; the other two-thirds are in the gap and are losing access to basic preventative services. The third consequence is that we are mortgaging our own future. Remember, that 40 percent of the poor in this country are children, and two thirds of them are in that gap. Also in that gap are tens of millions of young working Americans. These people constitute a large part of the shrinking workforce of tomorrow that we're expecting to fuel the economy and pay for a growing retired population. How are we going to do that in the face of $170 billion owed to foreign governments and nearly $3 trillion national debt. We are asking these people to be more productive than any other generation. We are asking them to do something that we have all refused to do, and that is to recognize that increases in personal consumption have to be balanced with increases in productivity.

There are also some disturbing professional implications. The first is the growing problem of uncompensated care that is catching physicians and providers between what society expects from our health care system and economic realities. Most of us do not understand the relationship between cost shifting and subsidizing care for the poor, and we don't understand the implications of taking cost shifting away from providers. The thought that a wealthy professional would be denying access to the poor in unacceptable to them. It puts us in a very vulnerable position politically. As the problems of the poor intensify, our state legislature will begin to react. They are going to say, "If you physicians are not going to take care of the poor voluntarily, we are going to force you to do that."

What can we do about this problem? To solve this crisis, we have to start by accepting three very hard realities. The first is that resources are limited, and that's a difficult reality. The second reality is that the rich are always going to have access to more health care than the poor. That's all right if what the poor receive is adequate and accessible. After all, one of the hallmarks of a capitalistic system is that goods and services are distributed on the basis of income, not necessarily on need or merit. But because of our system, our concept of universal access, we take for granted that the poor should have access to all health care services. The last reality, the inevitability of rationing. This is very difficult for physicians and providers to come to terms with. I suggest to you however, that rationing already exists in our system. We ration by income and transportation barriers. But more importantly, we ration through a lack of any policy to guide how we spend our health care dollars. We ration inadvertently by legislative decision. If you have a limited amount of money in the health care budget and you spend it on one thing, it's not available to be spent on something else.

We spend almost $2,000 per capita on health care in America. That is more than any other country in the world and yet our wellness as measured by morbidity and mortality statistics is not significantly higher than in England, which spends half as much. Why is that? Because we have no policy to guide how we spend our health care dollars. We are spending huge sums on some programs and none on other programs. We spend three billion dollars a year on neonatal intensive care while denying care to hundreds of thousands. We spend $50 billion a year on people in the last six months of their lives, while we are closing pediatric clinics. We are rationing by default. It's guided by no social policy, and it's not equitable. The reason we are rationing implicitly as opposed to explicitly is because we don't want to come to grips with our own limited resources.

To solve this problem of uncompensated care with all of the ominous implications for society and for physicians, we have to recognize that our health care system in indeed in flux. We have to build a new system that is based on the three realities that we've discussed: limited resources, acceptance of the fact that the rich will always be able to buy more health care than the poor, and that we're going to have to ration.

The government has a responsibility to pay for the poor, not for the elderly. The government should pay for the poor regardless of age. There is no reason Lee Iacocca needs Medicare. We should put an income eligibility requirement on it.

We must have a clear social policy, and we need to define adequate health. Then we need a universal insurance system to insure that people have access to that care. We need a clear social policy to guide that we spend those health care dollars in an efficient and equitable way. Should universal access to adequate health care be the first priority for spending additional dollars?

Once we get a definition of adequate health care, and we array our health care services on a priority basis, we are changing, in a very fundamental way, the nature of the rationing debate. We now shift the debate from the individual focus to a social focus. You are no longer debating which priority funding should be given or denied to which individual; you are debating which priority funding should be given to each service, given the reality of limited resources.

Society has made the decision to limit the amount of money it is spending on health care. Society needs to make the decision on how to spend the money. This would take the physicians out of the squeeze and they can again continue to be patient advocates.

But before we can do anything else, we must get a definition of adequate health care. I believe there are three steps that could assist in defining adequate health care. The first and probably most difficult would be to build a consensus. A group of dedicated providers and health experts would break down every dollar spent on that care. A list would be made of the number of people getting the service and the cost; the number of people not getting the service and the economic as well as health implications; and the cost to extend the service to cover everyone in giving them that service. Then what would it cost to extend other services to everybody in the unmet needs population. Arrange this list in a tentative priority order and present it at town hall meetings where citizens are actually getting involved in working through the trade-offs and choices necessary to set up a priority list of health care choices. Bring this information together and generate a final list that would be submitted to the legislature. You must, then, require that the funding go to the first item on your priority list for everyone in the population for whom the state has responsibility.

What this does is put accountability in the system. It no longer would be an abstract accounting exercise. It would mean the deletion of very specific services for very specific individuals off the bottom of the priority list. The debate then becomes far more focused.

The final point with this type of system is that, if it's done on the basis of sound clinical grounds, you can actually save money in the system.

What is the role of physicians in resolving this problem? The most significant role you have to play is to come to grips with your own limits. To recognize that health care resources in America are in fact limited. In addition, we need to adopt a policy, a statement on how to expend limited health care dollars—something that says the first priority is to extend the basic level of health care coverage. Then we can fight about the budget. But to do that, you must get involved in the definition of "adequate." Physicians are really the only group in this country that have the qualifications to provide sound clinical input to the state legislature. We need to say, "Yes, we are going to have to ration health care in this country. It is inappropriate and unethical for physicians to do it. Society needs to do it." Uncompensated care requires a partnership solution between public policymakers at the state legislative level and leadership in the medical community.

Raymond Schalow
Executive Director
Alaska State Medical Association

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Auxiliary News

Alaska State Medical Association Auxiliary

Dear Doctor:

Many members of the Alaska State Medical Association are unclear what their state auxiliary does. It is not a "social" organization, although we do hold social functions to raise money for projects.

The state auxiliary supports three projects during each year. It gives a $1,000 scholarship to an Alaskan student studying in a field related to medicine. During the holidays it collects contributions to the AMA-ERF through a holiday "sharing card" sent to each physician in Alaska. Each year there is a state health project. The past two years the auxiliary has supported the Alaska Lung Association's "Champ Camp," a camp for asthmatic children.

The Alaska State Medical Association Auxiliary is the liaison between the local auxiliaries and the national auxiliary. Presently Anchorage is the only local auxiliary in Alaska, but Fairbanks is in the process of forming one.

There is one meeting each year. It is held at the same time as the Alaska State Medical Association State Convention. The auxiliary state board meets throughout the year.

I hope this will give you a better understanding of what the ASMA Auxiliary does and that you will support our efforts.

Alice Samuelson
President, ASMA Auxiliary

Anchorage Medical Society Auxiliary

The Anchorage Medical Society Auxiliary will have a very busy year if all our plans come to fruition. We look forward to working with you this year on many of the issues facing our community.

To start out our year, we had a legislative workshop on September 17, 1988. This workshop was planned with education in mind and provided the opportunity to speak candidly with our legislators and candidates about our concerns. Speakers for the workshop were Senator Arliss Sturgulewski; Representative Sam Cotten; Representative Johnny Ellis; Lobbyist, Ashley Reed; David McGuire, M.D., and grassroots politician, Richard Nelson. We learned how to be most effective in our approach toward legislators, the seasons of the legislature, the importance of organization, how our legislative system really works from a successful lobbyist, and what things don't work in Juneau. Following the presentations, candidates and incumbents were invited for lunch and conversation with the participants.

All in all, I think the workshop was a success, and those people who attended felt they had spent their Saturday morning well.

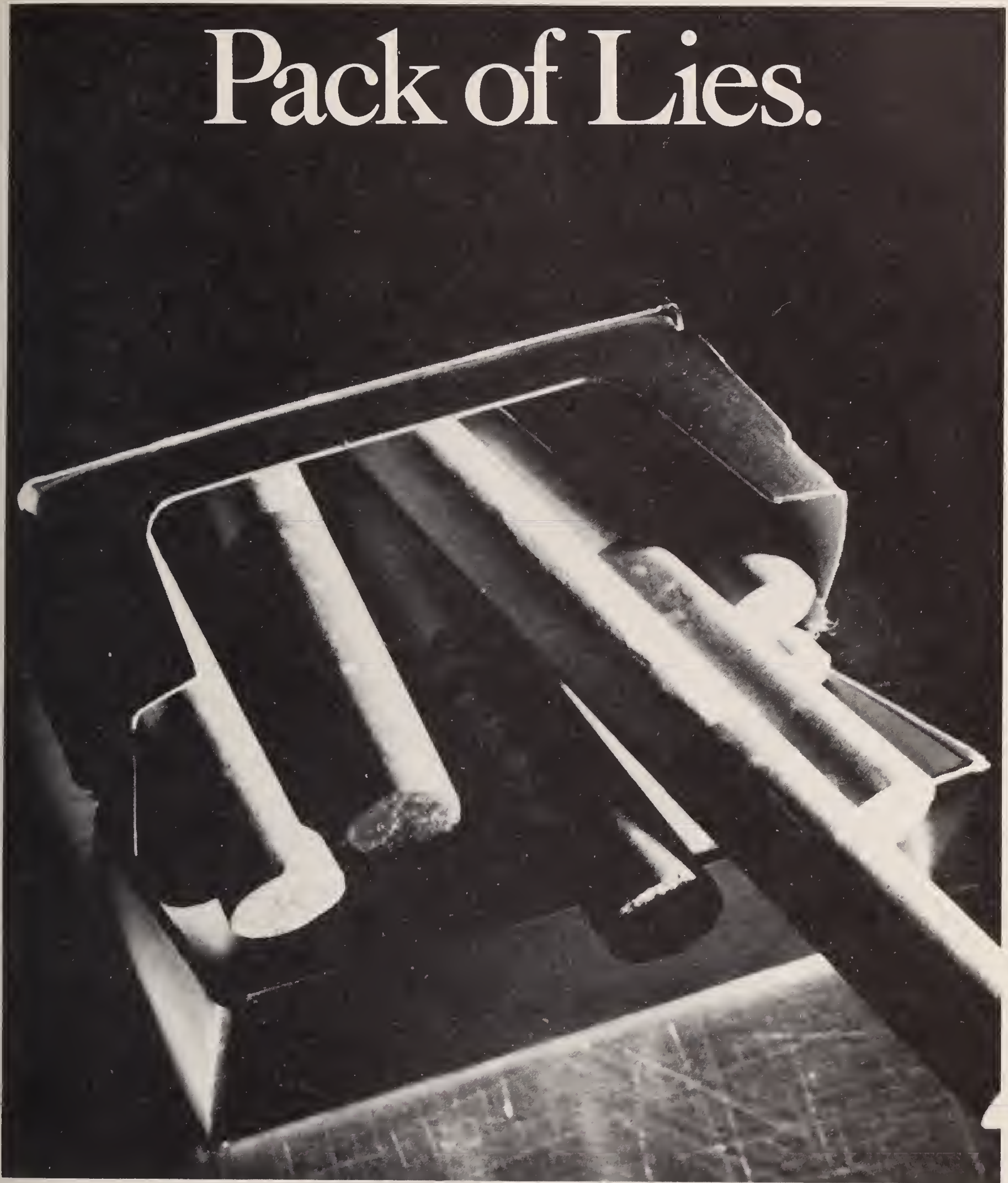
October 15, 1988 will be our fund raising effort for the year. A masked dance is planned to raise the estimated $2,000 needed for the health project budget this year. Tickets are now on sale for $15 each and can be reserved for you at the door by contacting me at 563-3028. YOUR TAX DEDUCTIBLE CONTRIBUTION WILL BE GREATLY APPRECIATED.

We have planned two health projects for the year. Our first project will be the Mid-Winter Blues workshop for the medical families in our community. This program will be presented to you in the November issue of *Heartbeat* and at the Anchorage Medical Society meeting in October. Tentative scheduling for this event is February 1989.

Our second project will be Children's Health Fair, usually held in April at the Northway Mall. If we can count numbers of excited children and parents, positive press coverage, and satisfied volunteers this is a highly successful community service project. So once again we will be asking you for help at the fair in individual stations, mock hospital, or testing sites.

Other projects the auxiliary is working on include volunteer help with the tort reform movement, seat belt legislation, and clothing donations to Covenant House youth shelter.

Lindsay Hansen Tucker
President, AMS Auxiliary Editorial



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Editorial

"Study on Cancer Causation in an Alaskan Village" by Julie E. Sprott in this issue of *Alaska Medicine* has important findings for all who are interested in improving the health of Alaskans. In the course of investigating a cancer cluster in an Alaskan village, Dr. Sprott uncovered a clear association and probable causal link to cigarette consumption. Surprisingly, although villagers were aware of the link between cigarette use and cancer, they held strong beliefs that the cancers occurring among village residents were due to other causes including fluoride, chlorine, radiation, and ozone-layer depletion.

Perhaps their beliefs should not be surprising after all, however, because the media have widely publicized cancer risks associated with the likes of fluoride, chlorine, radiation,and ozone-layer depletion. Unfortunately, the media message lacks adequate descriptions of the relative risk factors and even the *likelihoods* of causal associations between risk factors and cancer. Underscored by Dr. Sprott's findings is the inescapable conclusion that we have done an inadequate job in communicating effectively concepts of risk.

Consider recent events in Alaska. The widespread adverse effects of smoking are so well documented as to be irrefutable. In fact, the single most effective action that an individual can take to protect his or her health is never to start smoking; or if the person already smokes, to quit. The second most effective action that can be taken by an individual to protect his or her health is never to ride in a motor vehicle without buckling his/her seat belt.

In spite of overwhelming evidence that health risks from cigarette smoking and riding in motor vehicles without seat belts place individuals at risk of serious injury, illness, and death manyfold higher than exposure to fluoride, chlorine, radiation associated with fallout from atmospheric weapons testing in the 1950s, or depletion of the ozone layer, we have not used this knowledge effectively. It took years of painstaking efforts to enact legislation to restrict places where people can smoke and to increase taxes on cigarettes. Efforts to pass seat belt legislation have failed. One is reminded of an adage, "A thing that nobody believes cannot be proved too often."

It is with some dismay that we find that villagers attribute lung cancer to causes which pose little, if any, risk to health.

Our ability to understand risks and to base actions upon them is quite limited. Consider the following scenario: the mere rumor of the possible presence of a shark near a crowded beach on the east coast during the summer may precipitate a mass exodus characterized by hordes running to their vehicles, racing through congested streets without buckling their seat belts, piling out of their cars, flopping on the couch, turning on the television, lighting up a cigarette, and having a stiff drink while snacking on salt-laced munchies that contain large components of saturated fats.

We have obligations as health professionals to present to our patients and the public accurate and responsible medical information, both by our words and by our actions. We must remember these obligations whether we are dealing with AIDS, smoking, seat belt use, gun control, or alcohol and other drug abuse problems.

In order for us to be effective in communicating information about risk, we must first listen carefully to our patients so that we can understand their concerns and beliefs. We can then better help them to interpret the bewildering array of information in a way that can be useful to their own decision making about their lives and their health.

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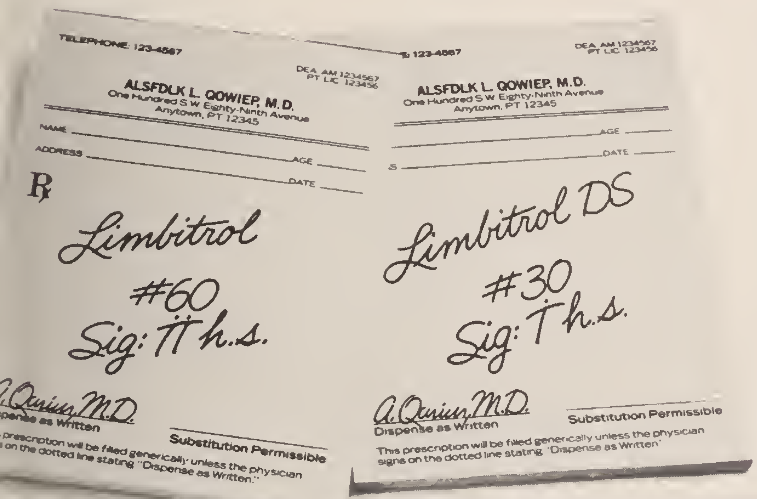

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Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants; concomitant use with MAOIs or within 14 days of monoamine oxidase inhibitors (then initiate cautiously, gradually increasing dosage until optimal response is achieved); during acute recovery phase following myocardial infarction.

Warnings: Use with caution in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur when used with anticholinergics. Closely supervise cardiovascular patients. Arrhythmias, sinus tachycardia, prolongation of conduction time, myocardial infarction and stroke reported with tricyclic antidepressants, especially in high doses. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving).

> ***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations. Consider possibility of pregnancy when instituting therapy.**

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: Use cautiously in patients with a history of seizures, in hyperthyroid patients, those on thyroid medication, patients with impaired renal or hepatic function. Because of suicidal ideation in depressed patients, do not permit easy access to large quantities of drug. Periodic liver function tests and blood counts recommended during prolonged treatment. Amitriptyline may block action of guanethidine or similar antihypertensives. When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady-state concentrations of the tricyclic drugs. Use of Limbitrol with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Should not be taken during the nursing period or by children under 12. In elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: Most frequent: drowsiness, dry mouth, constipation, blurred vision, dizziness, bloating. Less frequent: vivid dreams, impotence, tremor, confusion, nasal congestion. Rare: granulocytopenia, jaundice, hepatic dysfunction. Others: many symptoms associated with depression including anorexia, fatigue, weakness, restlessness, lethargy.

Adverse reactions not reported with Limbitrol but reported with one or both components or closely related drugs: *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke. *Psychiatric:* Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania, increased or decreased libido. *Neurologic:* Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns. *Anticholinergic:* Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue. *Endocrine:* Testicular swelling, gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Other:* Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. Withdrawal symptoms also reported with abrupt amitriptyline discontinuation. Therefore, after extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

Overdosage: Immediately hospitalize patient. Treat symptomatically and supportively. I.V. administration of 1 to 3 mg physostigmine salicylate may reverse symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

How Supplied: *Double strength (DS) Tablets,* white, film-coated, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt), and *Tablets,* blue, film-coated, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

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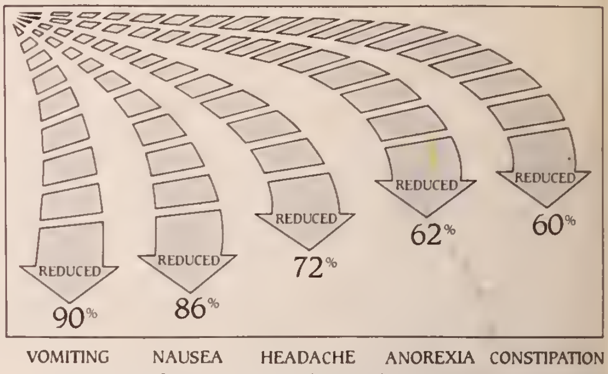
Caution patients about the combined effects of Limbitrol with alcohol or other CNS depressants and about activities requiring complete mental alertness, such as operating machinery or driving a car. In general, limit dosage to the lowest effective amount in elderly patients.

Percentage of Reduction in Individual Somatic Symptoms During First Week of Limbitrol Therapy*



*Patients often presented with more than one somatic symptom.

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Limbitrol® DS

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ALASKA MEDICINE

Journal of:

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Volume 30, Number 6 November, December 1988

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INDICATIONS AND USAGE

Cipro® is indicated for the treatment of infections caused by susceptible strains of the designated microorganisms in the conditions listed below

Lower Respiratory Infections caused by *Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter cloacae*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Haemophilus influenzae*, *Haemophilus parainfluenzae*, and *Streptococcus pneumoniae*

Skin and Skin Structure Infections caused by *Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter cloacae*, *Proteus mirabilis*, *Proteus vulgaris*, *Providencia stuartii*, *Morganella morganii*, *Citrobacter freundii*, *Pseudomonas aeruginosa*, *Staphylococcus aureus* (penicillinase and nonpenicillinase-producing strains), *Staphylococcus epidermidis*, and *Streptococcus pyogenes*

Bone and Joint Infections caused by *Enterobacter cloacae*, *Serratia marcescens*, and *Pseudomonas aeruginosa*

Urinary Tract Infections caused by *Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter cloacae*, *Serratia marcescens*, *Proteus mirabilis*, *Providencia rettgeri*, *Morganella morganii*, *Citrobacter diversus*, *Citrobacter freundii*, *Pseudomonas aeruginosa*, *Staphylococcus epidermidis*, and *Streptococcus faecalis*

Infectious Diarrhea caused by *Escherichia coli* (enterotoxigenic strains), *Campylobacter jejuni*, *Shigella flexneri*,* and *Shigella sonnei** when antibacterial therapy is indicated

*Efficacy for this organism in this organ system was studied in fewer than 10 infections

Appropriate culture and susceptibility tests should be performed before treatment in order to isolate and identify organisms causing infection and to determine their susceptibility to ciprofloxacin Therapy with Cipro® may be initiated before results of these tests are known, once results become available appropriate therapy should be continued As with other drugs, some strains of *Pseudomonas aeruginosa* may develop resistance fairly rapidly during treatment with ciprofloxacin Culture and susceptibility testing performed periodically during therapy will provide information not only on the therapeutic effect of the antimicrobial agent but also on the possible emergence of bacterial resistance

CONTRAINDICATIONS

A history of hypersensitivity to ciprofloxacin is a contraindication to its use A history of hypersensitivity to other quinolones may also contraindicate the use of ciprofloxacin

WARNINGS

CIPROFLOXACIN SHOULD NOT BE USED IN CHILDREN, ADOLESCENTS, OR PREGNANT WOMEN The oral administration of ciprofloxacin caused lameness in immature dogs Histopathological examination of the weight-bearing joints of these dogs revealed permanent lesions of the cartilage Related drugs such as nalidixic acid, cinoxacin, and norfloxacin also produced erosions of cartilage of weight-bearing joints and other signs of arthropathy in immature animals of various species (SEE ANIMAL PHARMACOLOGY SECTION IN FULL PRESCRIBING INFORMATION)

PRECAUTIONS

General: As with other quinolones, ciprofloxacin may cause central nervous system (CNS) stimulation, which may lead to tremor, restlessness, lightheadedness, confusion, and very rarely to hallucinations or convulsive seizures Therefore, ciprofloxacin should be used with caution in patients with known or suspected CNS disorders, such as severe cerebral arteriosclerosis or epilepsy, or other factors which predispose to seizures (SEE ADVERSE REACTIONS)

Quinolones may also cause anaphylactic reactions and cardiovascular collapse Anaphylactic reactions may require epinephrine and other emergency measures

Crystals of ciprofloxacin have been observed rarely in the urine of human subjects but more frequently in the urine of laboratory animals Crystalluria related to ciprofloxacin has been reported only rarely in man, because human urine is usually acidic. Patients receiving ciprofloxacin should be well hydrated, and alkalinity of the urine should be avoided The recommended daily dose should not be exceeded Alteration of the dosage regimen is necessary for patients with impairment of renal function (SEE DOSAGE AND ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION)

Drug Interactions: Concurrent administration of ciprofloxacin with theophylline may lead to elevated plasma concentrations of theophylline and prolongation of its elimination half-life This may result in increased risk of theophylline-related adverse reactions If concomitant use cannot be avoided, plasma levels of theophylline should be monitored and dosage adjustments made as appropriate

Antacids containing magnesium hydroxide or aluminum hydroxide may interfere with the absorption of ciprofloxacin, resulting in serum and urine levels lower than desired, concurrent administration of these agents with ciprofloxacin should be avoided

Probenecid interferes with the renal tubular secretion of ciprofloxacin and produces an increase in the level of ciprofloxacin in the serum This should be considered if patients are receiving both drugs concomitantly

As with other broad-spectrum antibiotics, prolonged use of ciprofloxacin may result in overgrowth of nonsusceptible organisms Repeated evaluation of the patient's condition and microbial susceptibility testing is essential If superinfection occurs during therapy, appropriate measures should be taken

Information for Patients: Patients should be advised that ciprofloxacin may be taken with or without meals The preferred time of dosing is two hours after a meal Patients should also be advised to drink fluids liberally and not take antacids containing magnesium or aluminum concomitantly or within two hours after dosing Ciprofloxacin may cause dizziness or lightheadedness therefore patients should know how they react to this drug before they operate an automobile or machinery or engage in activities requiring mental alertness or coordination

Carcinogenesis, Mutagenesis, Impairment of Fertility: Eight *in vitro* mutagenicity tests have been conducted with ciprofloxacin and the test results are listed below

- Salmonella/Microsome Test (Negative)
- *E coli* DNA Repair Assay (Negative)
- Mouse Lymphoma Cell Forward Mutation Assay (Positive)
- Chinese Hamster V_{79} Cell HGPRT Test (Negative)
- Syrian Hamster Embryo Cell Transformation Assay (Negative)
- *Saccharomyces cerevisiae* Point Mutation Assay (Negative)
- *Saccharomyces cerevisiae* Mitotic Crossover and Gene Conversion Assay (Negative)
- Rat Hepatocyte DNA Repair Assay (Positive)

Thus, two of the eight tests were positive, but the following three *in vivo* test systems gave negative results

- Rat Hepatocyte DNA Repair Assay
- Micronucleus Test (Mice)
- Dominant Lethal Test (Mice)

Long-term carcinogenicity studies in animals have not yet been completed

Pregnancy – Pregnancy Category C: Reproduction studies have been performed in rats and mice at doses up to six times the usual daily human dose and have revealed no evidence of impaired fertility or harm to the fetus due to ciprofloxacin In rabbits, as with most antimicrobial agents, ciprofloxacin (30 and 100 mg/kg orally) produced gastrointestinal disturbances resulting in maternal weight loss and an increased incidence of abortion No teratogenicity was observed at either dose After intravenous administration, at doses up to 20 mg/kg, no maternal toxicity was produced, and no embryotoxicity or teratogenicity was observed There are, however, no adequate and well-controlled studies in pregnant women SINCE CIPROFLOXACIN, LIKE OTHER DRUGS IN ITS CLASS, CAUSES ARTHROPATHY IN IMMATURE ANIMALS, IT SHOULD NOT BE USED IN PREGNANT WOMEN (SEE WARNINGS)

CONVENIENT *B.I.D.* DOSAGE

Recommended dosage schedule

Infection Site*	Severity of Infection	Dosage
Respiratory Tract* Bone and Joint* Skin/Skin Structure*	Mild/Moderate	500 mg q
	Severe/Complicated	750 mg q
Urinary Tract*	Mild/Moderate	250 mg c
	Severe/Complicated	500 mg c
Infectious Diarrhea*	Mild/Moderate/Severe	500 mg c

Nursing Mothers: It is not known whether ciprofloxacin is excreted in human milk, however, it is kno ciprofloxacin is excreted in the milk of lactating rats and that other drugs of this class are excreted in hum Because of this, and because of the potential for serious adverse reactions from ciprofloxacin in nursing ir decision should be made to discontinue nursing or to discontinue the drug, taking into account the impor the drug to the mother

Pediatric Use: Ciprofloxacin should not be used in children because it causes arthropathy in immature (SEE WARNINGS)

ADVERSE REACTIONS

Ciprofloxacin is generally well tolerated During clinical investigation, 2,799 patients received 2,868 co the drug Adverse events that were considered likely to be drug related occurred in 7.3% of courses, related in 9.2%, and remotely related in 3.0% Ciprofloxacin was discontinued because of an adverse 3.5% of courses, primarily involving the gastrointestinal system (1.5%), skin (0.6%), and central nervous (0.4%)

The most frequently reported events, drug related or not, were nausea (5.2%), diarrhea (2.3%), vomiting abdominal pain/discomfort (1.7%), headache (1.2%), restlessness (1.1%), and rash (1.1%)

Additional events that occurred in less than 1% of ciprofloxacin courses are listed below Those t quinolones are italicized

GASTROINTESTINAL *(See above)*, painful oral mucosa, oral candidiasis, dysphagia, intestinal per gastrointestinal bleeding

CENTRAL NERVOUS SYSTEM *(See above)*, *dizziness*, *lightheadedness*, *insomnia*, *nightmares*, *h tions*, *manic reaction*, *irritability*, *tremor*, *ataxia*, *convulsive seizures*, *lethargy*, *drowsiness*, *w malaise*, *anorexia*, *phobia*, *depersonalization*, *depression*, *paresthesia*

SKIN/HYPERSENSITIVITY *(See above)*, *pruritus*, *urticaria*, *photosensitivity*, *flushing*, *feve angioedema*, *edema of the face*, *neck*, *lips*, *conjunctivae or hands*, cutaneous candidiasis, hyperpi tion, erythema nodosum

Allergic reactions ranging from urticaria to anaphylactic reactions have been reported

SPECIAL SENSES *blurred vision*, *disturbed vision*, *(change in color perception*, *overbrightness o decreased visual acuity*, *diplopia*, *eye pain*, *tinnitus*, *bad taste*

MUSCULOSKELETAL *joint or back pain*, *joint stiffness*, achiness, neck or chest pain, flare-up of

RENAL/UROGENITAL *interstitial nephritis*, *renal failure*, polyuria, urinary retention, urethral vaginitis, acidosis

CARDIOVASCULAR palpitations, atrial flutter, ventricular ectopy, syncope, hypertension, angina myocardial infarction, cardiopulmonary arrest, cerebral thrombosis

RESPIRATORY epistaxis, laryngeal or pulmonary edema, hiccough, hemoptysis, dyspnea, bronch pulmonary embolism

Most of these events were described as only mild or moderate in severity, abated soon after the discontinued, and required no treatment

In several instances, nausea, vomiting, tremor, restlessness, agitation, or palpitations were ju investigators to be related to elevated plasma levels of theophylline possibly as a result of a drug interac ciprofloxacin

Adverse Laboratory Changes: Changes in laboratory parameters listed as adverse events without drug relationship

Hepatic – Elevations of ALT (SGPT) (1.9%), AST (SGOT) (1.7%), alkaline phosphatase (0.8%), LD serum bilirubin (0.3%)

Hematologic – eosinophilia (0.6%), leukopenia (0.4%), decreased blood platelets (0.1%), elevat platelets (0.1%), pancytopenia (0.1%)

Renal – Elevations of Serum creatinine (1.1%), BUN (0.9%)

CRYSTALLURIA, CYLINDRURIA, AND HEMATURIA HAVE BEEN REPORTED

Other changes occurring in less than 0.1% of courses were Elevation of serum gammaglutamyl tra elevation of serum amylase, reduction in blood glucose, elevated uric acid, decrease in hemoglobin bleeding diathesis, increase in blood monocytes, and leukocytosis

OVERDOSAGE

Information on overdosage in humans is not available In the event of acute overdosage, the stomach emptied by inducing vomiting or by gastric lavage The patient should be carefully observed and given s treatment Adequate hydration must be maintained In the event of serious toxic reactions from ov hemodialysis or peritoneal dialysis may aid in the removal of ciprofloxacin from the body, particular function is compromised

DOSAGE AND ADMINISTRATION

The usual adult dosage for patients with urinary tract infections is 250 mg every 12 hours For pati complicated infections caused by organisms not highly susceptible, 500 mg may be administered every

Respiratory tract infections, skin and skin structure infections, and bone and joint infections may with 500 mg every 12 hours For more severe or complicated infections, a dosage of 750 mg may be give hours

The recommended dosage for infectious diarrhea is 500 mg every 12 hours

In patients with renal impairment, some modification of dosage is recommended (SEE DOS ADMINISTRATION SECTION IN FULL PRESCRIBING INFORMATION)

HOW SUPPLIED

Cipro® (ciprofloxacin HCl/Miles) is available as tablets of 250 mg, 500 mg, and 750 mg in bottles of Unit-Dose packages of 100 (SEE FULL PRESCRIBING INFORMATION FOR COMPLETE INFORMATION)

***Due to susceptible strains of indicated pathogens. See indicated organisms in Prescribing Information.**

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ALASKA MEDICINE

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Volume 30 **November/December 1988** **Number 6**

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About the cover:
Walruses basking in the sun. Graciously furnished by John Hyde, Alaska Department of Fish and Game, Juneau, Alaska.

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The Use of Medicinal Plants by the Alaska Natives

by Robert Fortuine, M.D., M.P.H.

Photo of painting by Sandra J. Fongemie

Foreword

The creation of a new Institute for Circumpolar Health Studies at the University of Alaska by the Alaska State Legislature is a landmark in the history of medicine.

Senator Johne Binkley and his Senate colleagues, the principal sponsors of this Senate legislation, agreed with medical specialists from Alaska, the Soviet Union and Canada who testified before them that the severe health problems that plague our state are not endemic to Alaska alone, but to the whole circumpolar world. Each nation/state working alone has not been able to satisfactorily resolve problems related to nutrition, depression, alcoholism, and suicide and collectively acknowledge that it will only be through the collaborative efforts of all peoples in the North that any successful resolution and approach is to be found.

This issue of *Alaska Medicine*, along with the establishing of our Institute for Circumpolar Health Studies, are symbols of the positive union of the Alaska State Legislature with the University of Alaska, Alaska Department of Health and Social Services, Alaska Public Health Association, American Society for Circumpolar Health, and International Union for Circumpolar Health to institutionalize successfully the concerted efforts of all these professional groups to raise the standards of life for inhabitants of the North.

Heartfelt thanks are in order to all those who worked so very hard to make this dream a reality, including Dr. Robert Fortuine for the extensive work he gave to this publication. Special thanks go to Senators Johne Binkley, Jay Kerttula, Commissioner Myra Munson and their staffs, Governor Steve Cowper, the University of Alaska Board of Regents and President Donald D. O'Dowd, UAA Chancellor Donald Behrend, the House and Senate Health Sub-Committees, Mr. Jim Ayers, Ms. Mary Ellen Arnold, and Mr. Greg Pease.

Also, special thanks to Dr. Lydia Novak, President, Soviet Medical Workers Union and the Honorable J.D. Nicholson, Deputy Minister of Health for Canada who both came to Alaska to explore future relationships with us and who testified before the Alaska State Legislature. It is to all of these leaders that we extend our sincere thanks.

We appreciate all of your visionary efforts to improve the mental and physical health of all peoples of Alaska in the form of leaving the legacy of this Institute for future generations to work together in peace and for the good of all peoples of the North.

Ted Mala, M.D., M.P.H.
Director
Institute for Circumpolar Health Studies
University of Alaska Anchorage

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About the author . . .

Dr. Robert Fortuine recently retired from the U.S. Public Health Service after 26 years, all but three years spent with the Indian Health Service. He has served as director and part-time clinician in family medicine at Indian hospitals at Belcourt, North Dakota and Fort Defiance, Arizona; at Alaska Native Hospitals at Kanakanak and Bethel; and the Alaska Native Medical Center in Anchorage. From 1977 to 1980 he was posted to Geneva, Switzerland as International Health Attaché at the U.S. Mission to the United Nations (Europe), where he was liaison officer between the U.S. Government and the World Health Organization.

Dr. Fortuine received his M.D., C.M. degree at McGill, his M.P.H. at Harvard and is certified by the American Board of Preventive Medicine (Public Health). He is currently Vice President of the American Society for Circumpolar Health and serves as a member of the Council of the International Union for Circumpolar Health.

Dr. Fortuine has written numerous articles on the history of medicine in the Arctic regions, on such topics as traditional medicine, explorer–physicians, and the impact of disease on northern history. In addition, he is the author of: *The Health of the Eskimos, A Bibliography 1857-1967* (1968), *The Alaska Diary of Adelbert von Chamisso* (1986), *Alaska Native Medical Center. A History 1953–1983* (1986), and was the editor of *Circumpolar Health '84* (1985). In 1989, his book entitled *Chills and Fever: Health and Disease in the Early History of Alaska* will be published by the University of Alaska Press.

About the illustrator . . .

Sandra Fongemie, mother of three children, has been living and working in Alaska for 11 years. She is the owner and manager of the Alaska Herb and Tea Co. and spends most of her time gathering, drying and processing wild Alaska plants to sell as Alaskan teas.

The Use of Medicinal Plants by the Alaska Natives

by Robert Fortuine, M.D., M.P.H.

Illustrations by Sandra J. Fongemie

> *"The Lord created medicines from the earth, and a sensible man will not despise them."*
>
> —Ecclesiasticus 38:4

INTRODUCTION

In virtually all times, places, and cultures, man has employed plants and their extracts for healing. The earliest known medical treatise, the Ebers Papyrus from Egypt, is replete with descriptions of herbs and their therapeutic uses. Ancient Sumerian, Babylonian, Sanskrit, and Chinese texts provide further examples. In the *Iliad* Homer speaks of the skill of the Greek surgeons in treating battle wounds with herbs. A rich Greek, and later Roman, medical tradition followed that included Hippocrates, Galen, and Dioscorides, the latter the compiler of a vast catalogue of medicinal herbs in the first century A.D.

Nor is the use of healing plants limited to the great civilizations of the past that happened to have a written language to record their accomplishments. Traditional societies on every continent also have evolved empirical methods of healing using plant substances. In fact, it is not uncommon for totally divergent cultures to have hit upon the healing value of some of the same or closely related plants independently. The aboriginal peoples of Alaska were no exception.

In our modern era of attractively packaged and intensively marketed pharmaceuticals, with their detailed inserts containing scientific data on chemical structure and pharmacokinetics, we may easily forget how recently Western medicine depended on crude, non-standardized plant extracts laboriously prepared and compounded by a long-suffering apothecary. Of the common drugs used today, many came originally from such medicinal herbs, among them digitalis, quinine, morphine, ephedrine, atropine, reserpine, and curare. Furthermore, most of the earlier generations of antibiotics derived directly or indirectly from fungi or other plant substances.

The purpose of this study is to describe the use of medicinal plants by each of the principal Alaska Native cultures:—Aleut, Koniag, Chugach, Yupik and Inupiat Eskimo, Tlingit, Haida, Tsimshian, and the various subgroupings of the Athapaskans. Sources include the ethnographies and personal narratives of the Russian and early American periods, the more systematic ethnographies and ethnobotanical studies of the twentieth century, and finally some of the recent publications based on interviews with and observations of contemporary Alaska Native healers. This seeming wealth of material is misleading, however; first because of its having been collected over a two-hundred-year period, and second because the writers themselves are an odd collection of explorers, navigators, missionaries, naturalists, physicians, nurses, medical students, historians, anthropologists, botanists, and herbalists. Their medical and botanical knowledge, cultural sensitivity, professional bias, and scientific objectivity have varied enormously. Besides their differing qualifications, they have had varying depths of knowledge and varying abilities to communicate them to an "outsider." Some were practitioners themselves; others were self-designated carriers of the cultural tradition, without personal experience as a healer. Some of their accounts are fragmentary; others have resulted from a systematic review, using multiple informants, of the whole botanical realm of a defined area.

A further problem is the "contamination" of tradition, conscious or unconscious, which results from the influence of modern or "foreign" practices. For example, many traditional remedies known today are for diseases, such as syphilis and tuberculosis, that probably did not exist in aboriginal times. Likewise, some formulations use introduced wild plants, or non-traditional plant substances such as tea or flour. It is also likely that some Native practitioners have been influenced by a century or more of contact with Western medical practitioners, who at least in earlier times may have taught Native healers a few tricks of their own.

Still another problem is that nearly all of the source materials are local in scope, often within a subgroup of a larger cultural tradition. Plant lore may in fact have been even more local than this, since specific villages, families, and even individual practitioners probably developed their own methods of treatment, some of which were closely held secrets. The healing practices presented in this study may therefore not be widespread or even typical.

Much valuable information has also undoubtedly been lost for want of a chronicler. Relatively few early explorers, traders, or missionaries had the interest, language ability, intellectual curiosity, or patience to ferret out the necessary information. Healing practices sometimes died with their discoverers, or practitioners simply failed to pass on all their accumulated knowledge to their successors. In the past, as well as today, some practitioners with an important fund of knowledge on plant remedies may have been reluctant to share it with "outsiders," for fear of ridicule. Some treatments may have been hidden, and subsequently lost, in earlier times because they were associated with magical practices, or because they were the personal property of individual shamans, who themselves were under great pressure from the missionaries and others to cease their ministrations.

A particularly frustrating situation results from our inability to identify today some of the plants that were used, despite fairly detailed information on how they were collected, prepared, and administered.

Even with all these caveats and qualifications, however, much of value has survived about Alaska Native healing plants. I have tried to collect and analyze this information here in a form that will be useful to physicians, anthropologists, the general reader, and to the Alaska Native people themselves. Although this is by no means a botanical treatise, the information in the body of the paper is arranged according to the standard botanical classification of plants, as reflected in Eric Hultén *Flora of Alaska* (1968). To make the monograph more useful, I have provided a detailed index cross-referenced by the common and scientific names of plants, by their therapeutic use, and by Alaskan cultural groups.

THE PLACE OF MEDICINAL PLANTS IN THE HEALING TRADITION

Alaska's rigorous climate, short growing season, and extended snow cover ensured that the use of plant remedies could never become more than an adjunct to the healing tradition of the Alaska Native peoples. Further, the extent to which plants were actually employed in each culture depended largely on the geographical area, which in turn determined the diversity and seasonal availability of the local flora.

Healing with plants is part of the larger tradition of empirical medicine, which depends ultimately on the method of trial and error. The practice, which goes back to prehistoric times, is the direct ancestor of our own Western medical system. By no means restricted to plants, empirical medicine makes use of other healing substances, including animal products and inorganic materials. Beyond medicines, it encompasses surgery, massage, and the use of physical modalities such as heat, cold, and moisture.

In contrast to empirical medicine stands magico-religious medicine, which also has a very ancient tradition in nearly all cultures, and continues to have its proponents and practitioners today. This latter method, at least in earlier times, assumed that the cause of a serious disease or accident was supernatural, and could be attributed, for example, to sorcery, temporary loss of the soul, the intrusion of a magical object, or the breach of a taboo. The healer, usually known as a shaman or a medicine-man, achieved a cure by removing the cause of the disease, as he and the patient understood it, by means of magical methods such as charms, dancing, chanting, and trance-like states (Sigerist 1951, 191–213).

In Alaska both strands of traditional medicine were significant, but to differing degrees in the various cultures. In the north, for example, shamanistic healing held particular sway and the use of plant remedies was relatively unimportant, perhaps because plants were few in number and unavailable for much of the year. Among the Aleuts and Koniag both shamanism and plant remedies were important in healing, but in addition they developed a rich surgical tradition. In southeastern Alaska magical methods, including the magical use of plants for healing, were significant, whereas surgery seems to have played a minor role (Fortuine 1985b).

The individuals administering medicinal plants were of several types. Sometimes the sick person, or a family member, knew a remedy for the illness in question, and personally prepared the potion. Sometimes it was the shaman who prescribed, perhaps in conjunction with a healing ceremony, or at least a charm. Most often, however, herbal medicines were collected, prepared, and administered by a special village healer, sometimes called an herbalist, although he or she might also use non-plant substances. These individuals were usually older women, but not exclusively so. Both G. I. Davydov (1977, 177) and Yuri Lisianskii (Lisiansky 1814, 185) mention such healers in early nineteenth century Kodiak. Another early report, from southeastern Alaska, suggests that such healers did not enjoy a high reputation in the community, at least in that area (Khlebnikov 1976, 29). In more modern times female herbalists, as well as a few males, are known from several parts of Alaska (Juul 1979; Lantis 1959, 3; Preston 1961).

PREPARATION OF PLANT REMEDIES

The Alaska Natives for the most part used plants as "simples," that is, each plant was prepared in a certain way and administered by itself, without admixture of other ingredients. A specific plant, of course, might be used for several different symptoms or illnesses, but with a few exceptions the mixture of several substances, or "compounding," was not practiced. This usage is in striking contrast to that of the Western pharmaceutical tradition, which from ancient times until approximately a century ago was characterized by the simultaneous prescription of multiple ingredients, most of them of plant origin.

Alaska Native herbalists have used practically every part of some plants to prepare their medicines. The leaves, roots, and inner bark were probably the most popular sources of the active healing principles, but the buds, stems, fruits, flowers, seeds, pitch, sap, and wood also found their advocates. Often different parts of the same plant were prescribed for the same or different illnesses.

The most popular method for releasing the healing substance of a plant was by boiling it in water and drinking the resulting tea (sometimes also called an "infusion," or "decoction"). Only rarely was the boiled or roasted plant itself eaten, possibly because of the risk of excessive physiological effects. Sometimes fresh buds, tender stalks, or berries were eaten raw for their medicinal effect, but more commonly for their food value.

Externally, plant remedies were generally administered in the form of a poultice—a soft, moist, usually warm mass applied to a lesion of the skin. Poultices and other external applications took many forms, a common one being simply raw or cooked leaves. For some types of external diseases and injuries, the decoction made from boiling the plant was used for washing or bathing the affected area. Sometimes the leaves, roots, fruit, or other part of the plant were ground or mashed up and mixed with seal oil or fish oil to make a kind of ointment. Pitch was often warmed and applied to the skin as a protective layer. Some external preparations were primarily absorbant, such as "Alaska cotton," ashes, or spores.

Medicinal plants were used in still other ways. Pitch, roots, stalks, leaves, and other plant products were chewed for their topical effect on the mouth or throat. On occasion the plant was burned and the smoke inhaled, or it was heated on rocks, which were then sprinkled with water to produce a "medicated" steam. Certain plants were popular as switches to be used in the steambath.

A COMPENDIUM OF ALASKA NATIVE HEALING PLANTS

Algae

The medicinal use of seaweed and other algae in Alaska was apparently restricted to the peoples of the north Pacific rim. Available accounts, however, present a confused picture, since the types that were used are only identified by a common name.

The Aleuts used the oil from a kind of pond scum to apply to sore eyes and other inflamed areas (Bank 1953). Black seaweed, or laver (*Porphyra* spp.?) was employed by the Indians of southeastern Alaska to counteract nausea and to resolve blood clots. The Tsimshian applied black as well as yellow seaweed to cuts (McGregor). According to W. H. Evans and colleagues (*in* Smith 1973), a Tlingit remedy for stomach and bowel troubles, including dysentery, involved compressing *Porphyra umbicalis* into cakes and boiling them in water to make a tea. The Chugach Eskimos dried red seaweed, *Rhodoglossum latissimum*, and gave it to nursing mothers to promote milk flow.

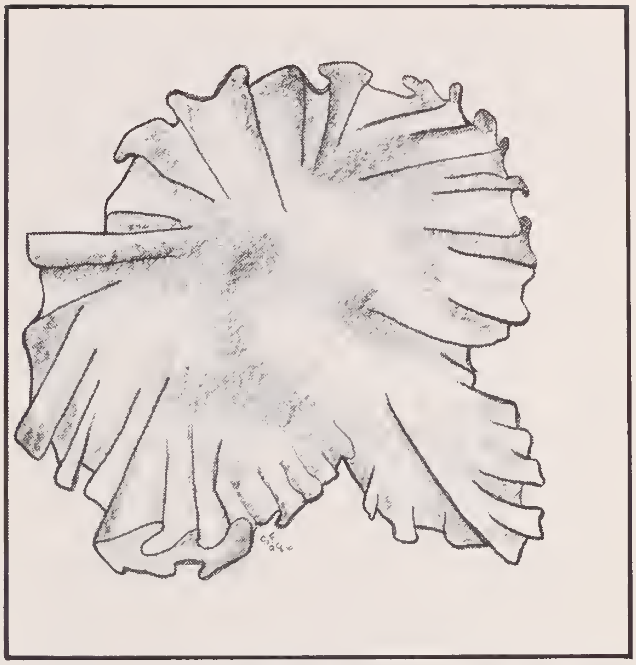

Figure 1. *Porphyra* spp.: red algae, laver, seaweed

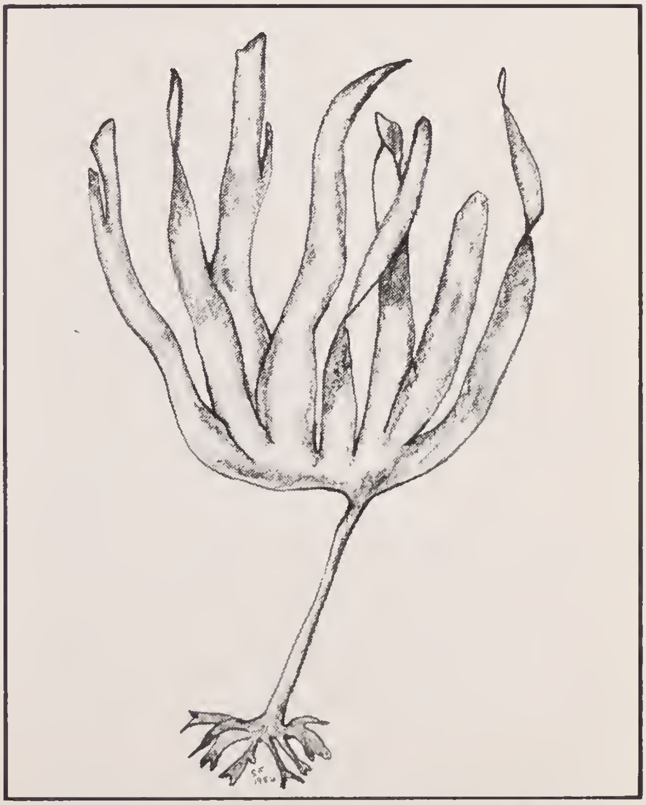

Figure 2. *Laminaria* spp.: brown algae, kelp

Among the Laminariaceae, which include the giant kelps, the Chugach applied "white seaweed" blades to soothe burning, itching feet. They also boiled *Agarum cribrosum* and drank the juice mixed with seal oil as a cold remedy (Wennekens 1985, 38–39). The Yakutat Tlingit applied warmed kelp pulp to swollen areas (De Laguna 1972, 656). Further south Dr. Eduard Blaschke (*in* Krause 1956, 284) in the mid-nineteenth century reported a headache treatment that involved warming the end of a giant kelp stem on a hot stone and allowing the steam to flow into the ear.

Fungi

In Europe surgeons have for centuries made use of the powder from "puff-balls" to control bleeding from wounds, while in America a similar practice was popular among the Pennsylvania Germans. The hemostatic properties of puff-balls were also widely known in American Indian groups (Vogel 1970, 224–225). The latter found other medicinal uses as well, including application to sores and scratches, burns, itchy rashes, earache, and headache. One tribe made a poultice by mixing the spores with the roots of the red baneberry and applying it to breast abscesses in nursing mothers (Burk 1983).

In Alaska fungi have also been used for healing, but for the most part the descriptions of them are so vague that exact identification is not possible. Puff-balls thought to be of the genus *Poria* (formerly *Fomes*) were used on the lower Kuskokwim. The Yupik applied the spores externally to burns, sores, and cuts, and boiled and ate the flesh for stomach-ache or constipation (Lantis 1959, 6). According to an early report the Koniag used what may have been a fungus to control bleeding from cuts (Black 1977). In Prince William Sound the people collected the mycelia of a certain fungus to serve as a wound dressing (Wennekens 1985, 39).

Lichens

Lichens have a simple plant structure consisting of a blue-green alga and a fungus living symbiotically. The so-called "reindeer moss," or *Cladonia rangiferina*, is probably the best-known example from the boreal regions. The Aleuts of Atka made an infusion from this lichen for the treatment of chest pain, and their hunters also ate the lichen when climbing a hill, in order to improve their wind (Bank 1953). Likewise, the Tanaina Athapaskans boiled and ate *Cladonia* to treat diarrhea (Kari 1981, 151), and the Upper Tanana band used an infusion to combat colds (McKennan 1959, 109).

In the lower Kuskokwim region the arctic kidney lichen (*Nephroma arcticum*) was boiled and eaten to counteract weakness (Oswalt 1957). The Chugach collected a lichen known as "old man's beard" (*Alectoria americana*) from spruce branches and piled it over a sick person in the steambath in order to hold in the heat, or to staunch bleeding from a wound (Wennekens 1985, 39–40). The Tlingit around Yakutat smashed another type of lichen, heated it on rocks with seal oil or goat tallow, and applied it to sores to aid healing. For cataract they gathered a green tree moss (*Parmelia* spp.?), mixed it with breast milk and made it into a pad for the eye (De Laguna 1972, 656–657).

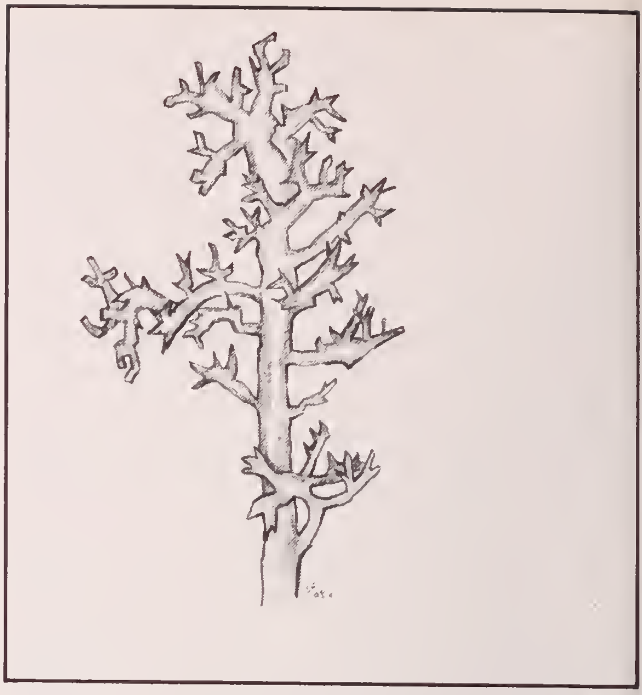

Figure 3. ***Cladonia rangiferina:*** **reindeer moss, caribou moss, lichen**

Sphagnidae (True mosses)

Among the true mosses are the sphagnum, or peat mosses, which are found in moist, acidic environments. In Europe they were widely employed as surgical dressings as late as the First World War. Many cultures have used them as menstrual pads or as diaper linings for infants (Viereck 1987, 65).

The Yupik Eskimos ate some kind of moss raw for the relief of diarrhea (Lantis 1959, 15), or for the treatment of postpartum bleeding (Ager and Ager 1980). Some soaked a soft yellow moss in seal oil and applied it to cuts (Lantis 1959, 19), whereas others used a kind of spongy moss soaked in seal oil as a means of controlling bleeding from a wound (Ager and Ager 1980). The Tanaina boiled a type of red sphagnum moss and held an injured or swollen part over the vapor. The same or a similar kind was warmed over hot rocks and applied as a poultice to sore eyes (Kari 1981, 147).

Equisetaceae (Horsetail family)

The horsetail family has only one genus, *Equisetum*. It is a rush-like flowerless plant with hollow, jointed stems and scale-like leaves. In Western culture it has been used externally since Roman times to control bleeding, as well as internally for the treatment of diarrhea and bladder disorders (Lewis and Elvin-Lewis 1977, 312, 340; Le Strange 1977, 109–110). Some American Indians boiled the stalks and washed lice-infested hair with the water. Others used extracts as a diuretic, for irregular menses, or as a wash for sore eyes. The plant contains a toxic substance, thiaminase, which is destroyed by heat (Viereck 1987, 37–38; Moerman 1977, 68–69).

In Alaska the Nelson Island Yupik dried and boiled the upper parts of *E. sylvaticum* and drank the bitter tea to control internal bleeding (Ager and Ager 1980). Around Prince William Sound the people mashed *E. arvense* to a paste and stuck it on the face with an eagle feather to treat pimples. After a few days the feather was removed along with the paste and, it was said, the pimples (Wennekens 1985, 40). The Tanaina burned the stem of some type of horsetail and applied the ashes to skin sores (Kari 1981, 75).

Lycopodiaceae (Club moss family)

Lycopodium species have a long healing tradition. Club moss was a sacred remedy of the Druids, for example, who employed it as a cathartic. The plant has had numerous other uses in medicine, for example as a diuretic, hypoglycemic agent, and tonic. The spores were dusted on skin diseases, and used by pharmacists to keep pills from sticking together. This preparation was listed in the *United States Pharmacopoeia* (USP) until 1950 (Millspaugh 1974, 730–731; Lewis and Elvin-Lewis 1977, 218). Among American Indians the spores were used as an anticoagulant and the plant itself as a purgative and antipyretic (Vogel 1970, 340; Moerman 1977, 112).

The Aleuts prepared an infusion from *Lycopodium clavatum,* the common club moss, to control postpartum pain (Bank 1953). The Tanaina Indians boiled a similar species, cooled the water, and applied it externally for headache or sore eyes (Kari 1981, 136).

Athyriaceae (Lady fern family)

Three genera of lady ferns occur in Alaska, widely distributed over the southern half of the territory. The only reported medical application was by the Lime Village Tanaina, who boiled *Athyrium filix-femina* for use in kidney trouble or asthma (Kari 1981, 108).

Blechnaceae (Deer fern family)

The deer ferns are represented by a single species in Alaska, *Blechnum spicant*, which is found along forested shores on the Pacific rim. The Chugach used it as a switch in the steambath. They also considered it a medicine for the whole body and attributed to it, among other properties, the ability to keep sores from breaking out (Wennekens 1985, 41).

Aspleniaceae (Spleenwort family)

Two species of the spleenwort ferns (genus *Asplenia*) are found in Alaska in crevices and rock ledges, chiefly in the southeast. The sole medicinal use was reported by Dr. Blaschke (*in* Krause 1956, 283), who noted that the Tlingit prepared an infusion from it for chest inflammations due to "catarrhs," or colds.

Aspidiaceae (Shield fern family)

The family of shield ferns has had several important medical uses. The so-called "male fern" (*Dryopteris felix-mas*) was in the USP from 1831 to 1965 as a tapeworm remedy, under the name Aspidium oleoresin. Among American Indians *Dryopteris* was employed as a tea for stomach trouble and as an antidote for shellfish poisoning (Vogel 1970, 304; Moerman 1977, 64–65).

In Alaska the shield ferns, which grow in woods and moist places, also had a few medicinal applications. The Indians around Lime Village boiled *D. dilatata* (also known as *D. austraica*) to make an infusion that they drank for kidney trouble or asthma, or used as a soothing eyewash (Kari 1981, 107–108). The Eskimos of Nunivak Island drank the tea for stomach-ache (Lantis 1959, 5).

Pinaceae (Pine family)

Members of this large and widely distributed family of coniferous trees have had medicinal uses among Western cultures since ancient times. Turpentine was prepared from several species, as were various resins, pitches, and balsams. The members of the pine family have been particularly esteemed for their expectorant effect (Lewis and Elvin-Lewis 1977, 301). The bark of the spruce is said to be diaphoretic, and the inner bark was the constituent of infusions for bowel complaints and arthritis. The powdered resin was sprinkled over wounds to stimulate tissue growth (Millspaugh 1974, 654–655).

Both pines and spruces were widely used internally by the American Indians as diuretics, laxatives, antirheumatics, and for respiratory illnesses and venereal disease. Many were also esteemed for their external effect on skin disorders and wounds (Vogel 1970, 346–347; Moerman 1977, 137–141). A kind of Indian spruce beer made of cones and twigs boiled in maple syrup was recognized by early settlers as a powerful antiscorbutic (Millspaugh 1974, 655).

In Alaska the pine family is represented by two species of pine, a single species of tamarack, three species of spruce, two species of hemlock, and two species of fir.

Blaschke (*in* Krause 1956, 284) reported that the juice of *Pinus canadensis* mixed with tallow from the mountain goat was a Tlingit remedy for diarrhea. Although exact identification is uncertain, he may have been referring to *P. contorta*, or lodgepole pine, which is common throughout southeastern Alaska (Viereck and Little 1986, 47). The sap of the latter was used as an internal remedy for coughs and colds by some Alaska Native groups (R. F. Taylor *in* Smith 1973). Blaschke (*in* Krause 1956, 284) also mentions a Tlingit medicine for syphilis consisting of an infusion of the sprouts and bark of another uncertain type of pine he calls *P. inops*, mixed with *Ledum palustre* (Labrador tea) and a species of cedar. The Haida sometimes applied a kind of pine-tree gum as a poultice to wounds (Niblack 1888, 349).

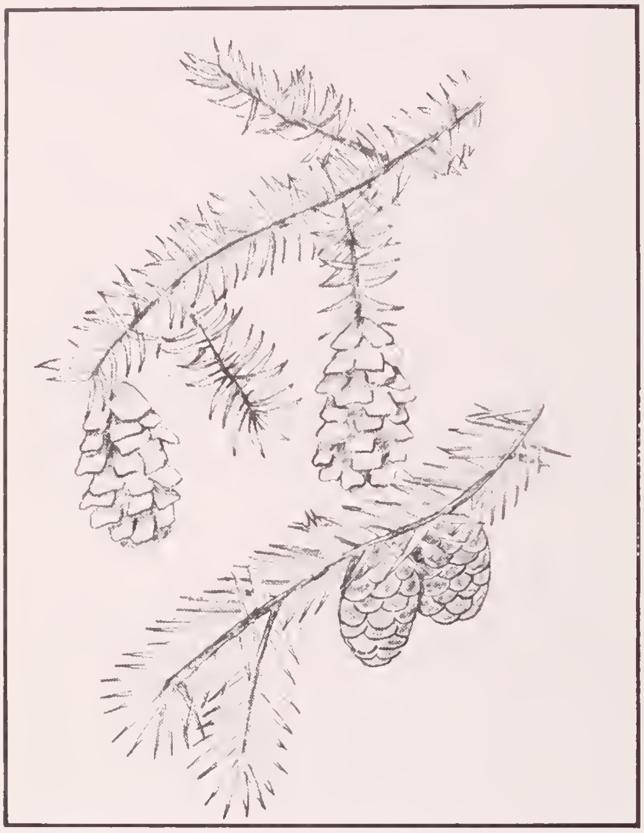

Figure 4. ***Picea glauca:*** **white spruce, Canadian spruce;** ***Picea mariana:*** **black spruce, bog spruce**

Products of the various spruce trees (*Picea* spp.) had many medicinal uses in Alaska. The Eskimos around Norton Sound applied the resin of the white spruce, *Picea glauca* (or *P. canadensis*), to wounds (Anderson 1939). Those around Kotzebue Sound recommended chewing spruce gum on the same side of the mouth as a partial facial paralysis (Bell's palsy?). They also prepared an infusion from the needles, which they took for chest colds or as an all-purpose remedy. Inhaling the smoke from the burning needles was thought to help prevent the spread of influenza during an epidemic (Mauneluk Cultural Heritage Program 1976, 14, 27, 41). On the Kuskokwim the Yupik chewed the needles or made an infusion from them for cough (Lantis 1959, 24). The Indians around Cook Inlet chewed the gum as a treatment for tuberculosis or applied it externally to minor cuts (Osgood 1937, 117). The Athapaskans of the Koyukuk River boiled white spruce needles and drank the infusion for kidney problems or simply to promote good health. Externally they applied the tea to sores or dry skin. The pitch was thought to promote healing in infected cuts and skin sores (Nelson 1983, 49–50).

The Chugach found many uses for the Sitka spruce, *P. sitchensis.* They prepared a tea from the bark or from the soft young branch tips for colds, cough, or tuberculosis, and chewed the pitch to relieve a sore throat, or spread it on cuts. The crumbly wood from a decayed spruce stump could be warmed on a hot rock and poured into an aching ear. Whether warmed or not it could be sprinkled on an infected cut or boil (Wennekens 1985, 41–42).

The Tanaina Indians had multiple uses for what Kari (1981, 1–5) calls the "jack spruce" (all three Alaskan species occur in the area). They made the white inner bark into a tea that was said to be good for heart disease, kidney trouble, ulcers, stomach-ache, colds, sore throat, mouth sores, or even tuberculosis. They found the same inner bark, glued on with pitch, useful as an external application on cuts, burns, and sores. A foreign body in the eye could sometimes be removed by touching it lightly with a sliver of the bark. The soft, white pitch inside the wood served as a salve for cuts, sores, and infections. The spring sap was taken internally for tuberculosis, or smeared on cuts and burns. They chewed spruce pitch gum for heart trouble or tuberculosis, and also applied it to cuts to control bleeding. For sore eyes they bit the root and allowed the juice to drip into the eyes. The needles were made into a tea that served as a cough medicine and emetic, and a similar infusion made by boiling the top of the tree became a remedy for aches, colds, and tuberculosis. Finally, an ingenious use was to shake repeatedly the powder from a spruce cone into a draining ear. The purulent discharge gradually collected into a firm mass that then could be removed from the ear canal.

The Ingalik of the lower Yukon River scraped off and heated the gum from an unspecified type of spruce for application to burns, and boiled down the inner bark as either an internal or external remedy for sore throat (Osgood 1958, 229–230). Han midwives, on the upper Yukon, smeared spruce gum and charcoal on the navel stump after childbirth (Osgood 1971, 46). The Kutchin applied grease, then spruce gum, on eyes afflicted with snowblindness (Osgood 1936, 93). They also boiled the pitch and spread it on sores, or took it internally as a treatment for tuberculosis (Nelson 1973, 37). Boiled old spruce pitch was also a favorite internal remedy of the Upper Tanana Indians, who also spread the fresh pitch on cuts to prevent infection (McKennan 1959, 109).

Other Athapaskans boiled spruce needles and washed skin rashes with the liquid. An infusion of spruce needles was taken internally as a cold remedy, or as a treatment for urinary infections. The latter were also sometimes treated by sitting in a dilute solution of boiled needles. Various other Athapaskan bands had multiple external uses for spruce pitch, which was usually warmed to soften it before application. Among other conditions treated were infected cuts, sores, headache, backache, warts, and "blood poisoning" (Hall 1979).

The Nunamiut Eskimos of the northern interior applied spruce gum to cuts to prevent infection (Gubser 1965, 214). The Chugach ground up the whole root to a fine powder, cooked it for three days, and administered it for generalized illnesses. They also used hot pitch, presumably of the spruce tree, as a treatment for frostbite (Birket-Smith 1953, 116–117). An early nineteenth-century report from Kodiak Island described the application of what may have been ground-up spruce stump to control bleeding (Gedeon *in* Pierce 1978, 129). In southeastern Alaska the Yakutat Tlingit drank a spruce bark infusion as a cough remedy (De Laguna 1972, 657), whereas the Tsimshian prepared an infusion from the young boughs for use as a laxative (McGregor 1981).

For tuberculosis the Indians around Yakutat employed an infusion made from the bark and pitch of a hemlock tree not otherwise identified (De Laguna 1972, 657). The Tsimshian warmed hemlock pitch in hooligan (eulachon) oil and applied it to eczema, or smeared it unmixed on sores (McGregor 1981).

Cupressaceae (Cypress family)

The cypress family of evergreens includes the junipers, cedars, and cypresses. Juniper probably had medicinal use even among the ancient Assyrians, and likewise cedar products were known to have been employed by the Egyptians. Over the past several hundred years in Europe, juniper berries were prescribed for their stimulant and diuretic properties (Wheelwright 1974, 51, 59, 196), and were sometimes burned in the sick room to purify the air (Le Strange 1977, 215). The berries are the source of juniper oil, which has long been esteemed as a diuretic (*Merck index* 1983, 756) and antiflatulence remedy (Reynolds and Prasad 1982, 676). Juniper tar (USP), which is thought to be largely irritant and anti-eczematous, has been in recent times a component of ointments, shampoos, and emulsions (*Merck index* 1983, 756). The plant is also a source of podophyllotoxin, which has some antineoplastic effect. A type of cedar wood contains thujaplicins, which have antifungal properties (Lewis and Elvin-Lewis 1977, 133, 360).

American Indians found numerous medicinal uses for the needles, berries, and oil of juniper and of the cedar tree, particularly for respiratory diseases, dysentery, kidney trouble, and venereal disease (Vogel 1970, 290; Moerman 1977, 98–102).

In Alaska this family is represented by the red cedar, Alaska cypress, and two species of juniper.

As already mentioned, Dr. Blaschke (*in* Krause 1956, 284) described a syphilis remedy which included a cedar *Thuja excelsis* (*T. plicata?*), in addition to Labrador tea and a type of pine. In Prince William Sound the Natives applied the charcoal of yellow cedar, *Chamaecyparis nootkatensis* (called Alaska cypress by Hultén [1968, 65]), to burns in order to prevent scarring. The ashes were considered helpful for pimples and boils (Wennekens 1985, 42).

Figure 5. *Juniperus communis:* common mountain juniper

Juniperus communis, the common mountain juniper, found several medicinal uses among the Alaska Natives, principally among the Athapaskans. The Chandalar Kutchin ate the berries for chest pain (McKennan 1965, 71) and along the Porcupine River the local Kutchin drank a tea made from the berries and boughs for colds and other aches and pains (Nelson 1973, 41). The Tanaina boiled the branches into a tea that was prescribed for colds, sore throat, urinary retention, and tuberculosis. Smoke from the burning needles was also thought to be useful for colds (Kari 1981, 11). The Indians of the Upper Tanana River prepared an infusion from juniper sprigs for tuberculosis, raw gums, or toothache, and they ate the berries raw or cooked for internal pains (McKennan 1959, 109). The Eskimos of the Kotzebue Sound area ate the berries and needles raw, or prepared a tea from them for the relief of colds, influenza, arthritis, gall bladder disease, or kidney troubles (Jones 1983, 110; Mauneluk Cultural Heritage Program 1976, 12-13, 28).

Gramineae (Grass family)

The botanical classification of grasses is very complex and identification of individual species is difficult. Several contain as yet undefined antibiotic principles (Lewis and Elvin-Lewis 1977, 364). The American Indians had medicinal uses for a number of individual grasses, for example as diuretics, emetics, analgesics, antipyretics, tonics, and eyewash (Moerman 1977, 338).

Hultén (1968, 82-197) has listed no fewer than 52 genera and 191 species of grasses, not to mention many subspecies, that may be found in Alaska and the surrounding area. None of those used by the Alaska Natives for medicinal purposes has been precisely identified. Hieromonk Gedeon described the Koniag using a grass called *amagot'*, which they crushed, boiled, and drank twice a day in the form of a tea for venereal disease, especially when it was complicated by sore throat. Another type of grass, called *shulialiunak*, was collected near the village of Karluk and also used for venereal disease. The Natives boiled it in a sealed dish and took a little of the infusion along with a root called *mzhulingok*, which was chewed (*in* Pierce 1978, 130; Black 1977). The Chugach employed grass to tie the umbilical cord after childbirth (Birket-Smith 1953, 84). Finally, the Tanaina Indians were said to chew a certain grass and apply it to painful bee stings (Kari 1981, 77).

Cyperaceae (Sedge family)

The sedges are a large family of rush-like plants which resemble the grasses but have solid stems. Some types were found helpful by the American Indians to treat stomach troubles and snake-bite, or to induce vomiting (Moerman 1977, 333).

Hultén (1968, 197-280) has described eight genera and 144 species from Alaska and the surrounding territories. The cotton balls of *Eriophorum russeolum*, or "Alaska cotton," were used by the Yupik Eskimos of the lower Kuskokwim and on Nunivak Island to absorb the discharge from draining boils, and sore, inflamed eyes (Lantis 1959, 17; 1946, 202). The stems and leaves of the tall cotton grass, *E. angustifolium*, were sometimes eaten raw to improve the general health (Oswalt 1957, 27).

Araceae (Arum family)

The members of the arum family bear flowers on a fleshy spike surrounded by a hood-like leaf. They have been used for medicinal purposes in Western civilization at least since the fourth century B.C. (Wheelwright 1974, 78). The corm was listed in the USP until 1873 as a stimulant, expectorant, and diaphoretic. The corms have an extremely acrid taste that causes nausea and vomiting. American Indians discovered that the poisonous principle could be dissipated by heat. They used the plants primarily for disorders of the bowels or chest, and as a laxative and stimulant (Vogel 1970, 321–322). A *Lysichiton* species was employed as a sedative, antirheumatic, and dermatological preparation (Moerman 1977, 302).

In Alaska the arum family has three representative species, one of which was used medicinally by the Natives. *Lysichiton americanum*, or yellow skunk cabbage, is widely distributed in Prince William Sound and southeastern Alaska. The Tlingit of the Yakutat region cooked and crushed the root shavings and applied them mixed with seal oil or goat tallow to infected wounds, bruises, or swellings. They also boiled the root with water and seal oil and drank the mixture to treat lung trouble. Sometimes they allowed the concoction to dry and then scraped and inhaled the odor (De Laguna 1972, 655–657; Evans *in* Smith 1973). The Chugach also used skunk cabbage roots for healing in earlier times, but details are not available (Wennekens 1985, 43). Some Athapaskans boiled the plant and drank the tea for the relief of colds (Hall 1979).

Liliaceae (Lily family)

The Liliaceae are a large family of shrubs or woody plants springing from bulbs or tubers, several of which are edible. Plants of the genus *Helleborus* have been used medicinally since the time of Hippocrates. The genus *Veratrum*, or false (green) hellebore, is native to America and has also been an important medicinal herb for centuries because of its ability to depress heart action, lower blood pressure, and treat arthritis (Wheelwright 1974, 163; Millspaugh 1974, 710). These pharmacological effects are due to ester alkaloids, including jervine, which in excessive quantities can be a potent

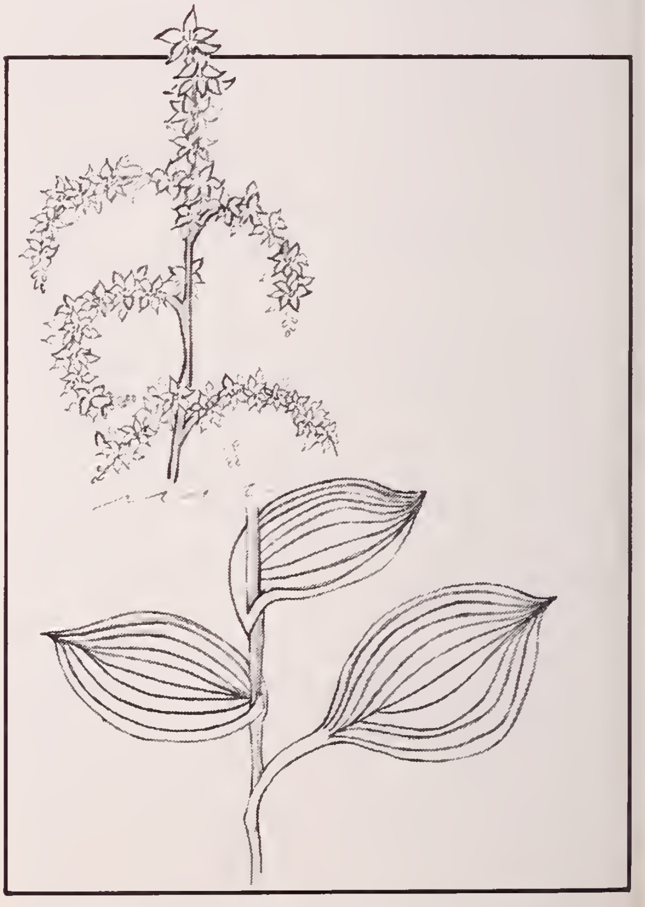

Figure 6. ***Veratrum viride:*** **false hellebore, skookum root**

poison, causing vomiting, purging, excessive salivation, and ultimately asphyxia (Hultén 1968, 306; Heller 1981, 85). Several prescription medications containing veratrum are still on the market for treating hypertension (Billups and Billups 1987, 650).

As early as the seventeenth century there is a report of the false hellebore being used by the Indians to "cure wounds." Some tribes have employed it for colds, internal pains, constipation, or they made an ointment from it for skin troubles (Vogel 1970, 315–316; Moerman 1977, 198–199).

Although 12 genera and 19 species of the lily family occur in Alaska, the only reported member used medicinally was the false hellebore, which is found principally in the Cook Inlet region and southeastern Alaska. *Veratrum viride* is known locally to the Tlingit as "skookum root," and because of its physiological effects, was considered "strong medicine."

Around Yakutat the Indians dried the leaves and roots, then boiled them and added oil to make a remedy for menstrual cramps and pneumonia (De Laguna 1972, 659). The Haida used the root as a painkiller and tranquilizer (McGregor 1981). The Tanaina Indians employed an infusion made from the roots as a powerful emetic.

The external uses of the plant were more numerous and no doubt safer. On Kodiak Island the people sometimes burned the stem near the root, mixed it with mother's milk to form a poultice, and applied it to the eye as a treatment for cataract (Preston 1961). The Tanainas applied the raw root directly to a painful tooth, or burned the root to ashes, then smeared them on a painful area. A third method involved soaking the root in water, mashing it up, and applying either the root itself, or the liquid, under a bandage for sores, cuts, rashes, or "blood poisoning" (Kari 1981, 91–92). In southeastern Alaska an early report describes the Tlingit stuffing veratrum plants directly into the nose to treat a cold (Swanton [1908], 446). More recently a snuff made from the veratrum root was described as helpful for sinus trouble, since it induced powerful sneezing (McGregor 1981). The root was also ground up, mixed with seal oil, and applied directly to the scalp to counteract baldness or dandruff (De Laguna 1972, 658). The Tsimshian treated insanity by rubbing the root directly on the head. Although the leaves were not commonly used, one report mentions applying them to arthritic joints (McGregor 1981).

Iridaceae (Iris family)

The iris family includes, in addition to the plant of that name, the gladiolus, crocus, and other common flowers. Medicinal use of the genus *Iris* dates back at least to Theophrastus in 300 B.C. (Le Strange 1977, 150). Members of the genus have emetic and cathartic properties and are the source of substance known as orris, which is still used in tooth powders, at least in Great Britain (Reynolds and Prasad 1982, 680). The dried root of *I. versicolor* was listed in the *National Formulary (NF)* until 1942, for use as a tonic, diuretic, or purgative. The genus *Iris* was widely used as a medicine by the Indians of North America. Eighteenth century accounts describe it as an emetic, cathartic, or as an external application for leg sores (Vogel 1970, 284). Other tribes used *Iris* for venereal disease, stomach problems, or rheumatism (Moerman 1977, 96–97).

The iris family is represented in Alaska by two genera and three species. Only one of these, *I. setosa*, or wild flag, seems to have been used medicinally. The Aleuts of Unalaska considered an infusion from the roots helpful as a laxative (Bank 1953). The Tanaina also used the plant in earlier times, but the details are no longer known (Kari 1981, 81). Around Angoon the Tlingit were said to prepare a root tea for use as a cathartic, emetic, diuretic, and stimulant (Viereck 1987, 40).

Salicaceae (Willow family)

The willow family of shrubs and trees has but two genera, *Salix*, or willow proper, and *Populus*, or poplar (cottonwood, or aspen). The antipyretic property of willow bark has been known at least since the time of Dioscorides (first century A.D.). The active principle, salicin, was isolated in 1827, and also occurs in the bark of poplar trees. With later purification and chemical modification, acetyl salicylic acid, or aspirin, was synthesized in 1899. In earlier times the bark extract was used to treat intermittent fevers, rheumatic fever, and gout (Goodman and Gilman 1970, 314). A chemical derivative, salicylic acid, is widely used as a keratolytic and mild disinfectant, and is even said to have some antibacterial and antifungal properties (Lewis and Elvin–Lewis 1977, 151). American Indians used willow products for fevers, colds, asthma, diarrhea, colic, sore eyes, venereal disease, worms,backache, sore eyes, and various types of stomach troubles (Vogel 1970, 392–393; Moerman 1977, 378–379).

Extracts of the genus *Populus* were used in much the same way as those of *Salix*, although the physiological effects were generally less pronounced. A substance called trichocarpin, with some antifungal effect, has been derived from the bark of poplar trees, and a chemical with antituberculosis effects (bisabolol) has been isolated from young shoots (Lewis and Elvin-Lewis 1977, 360). A kind of "balm of Gilead" was made from the resinous winter buds boiled in tallow, and used by certain Indian groups for colds and bronchitis (Lewis and Elvin-Lewis 1977, 308).

This family is represented in Alaska by both genera. *Salix* shows great variability here, as elsewhere; Hultén (1968, 333-363) lists some 56 species or varieties of willow found in Alaska.

The balsam poplar, or cottonwood (*P. balsamifera*),

Figure 7. ***Populus balsamifera:*** **cottonwood, balsam popular**

Figure 8. ***Salix pulchra:*** **diamondleaf willow**

was used for medicinal purposes primarily by the Athapaskans. The Ingalik prepared an infusion from the outer bark as a remedy for stomach-ache (Osgood 1958, 230). The Upper Tanana band, on the other hand, made their infusion from the buds and administered it for colds and internal ailments (McKennan 1959, 109). The Tanaina found the juice from the inner bark helpful as a general tonic late in the spring, and also inhaled the smoke from burning winter branches to soothe cold symptoms. For external use they dried the winter buds, mashed them to a powder, and mixed them with oil as a salve for sores, rash (especially diaper rash), and frostbite (Kari 1981, 16-17). Some Athapaskans boiled cottonwood leaves in a pan and drank a small amount of the liquid for respiratory infections (Hall 1979). The Eskimos around Kotzebue Sound ate the cambium layer of young cottonwood as a laxative (Mauneluk Cultural Heritage Program 1976, 27).

Recorded use of the willow in medicine was particularly in the domain of the Yupik Eskimos. The Nunivak Islanders chewed the leaves of *S. fuscescens* or *S. pulchra* for a sore mouth (Lantis 1946, 202), and applied the willow catkins to sore, watery eyes (Lantis 1959, 60). *S. arbusculoides* bark was employed as a poultice for sores (Oswalt 1957, 30). On Nelson Island the people used *S. planifolia* (or *S. phylicifolia*), also known as the ptarmigan willow, for sores, or watery eyes (Ager and Ager 1980).

Other types of willow, not further identified, also found medicinal application in southwestern Alaska. Absorbant willow catkins were applied to cuts. On the Kuskokwim an infusion prepared from the leaves and bark was administered for hemoptysis, and the inner and outer bark were boiled and the liquid used as a gargle for sore throat or mouth sores. Others shredded the inner bark and applied it as a poultice to open sores (Lantis 1959, 5-6, 19, 23).

The Nunamiut of the northern interior sometimes chewed the green bark to relieve backache (Gubser 1965, 240), whereas the people of Prince William Sound chewed the cambium layer for generalized pain (Wennekens 1985, 43). Eskimos and some Athapaskans chewed willow leaves into a paste and applied it on hornet or bee stings (Preston 1961; Mauneluk Cultural Heritage Program 1976, 30, 40; Hall 1979). The Ingalik boiled together the juice from willow, cottonwood, and alder, and administered it for excessive menstrual bleeding (Osgood 1958, 230). The Tanaina peeled off the bark from a new growth and chewed the inner substance as a remedy for stomach-ache or headache (Kari 1981, 23). Other Athapaskans boiled willow branches and bathed in the water to treat skin infections (Hall 1979).

Myricaceae (Wax myrtle family)

The wax myrtle family is made up of shrubs or small trees, including a few which have been employed as medicines. The wax myrtle itself, sometimes called bayberry, was a common remedy in India, where it was used as a tonic, and, when mixed with other ingredients, as a remedy for dysentery and chills (Wheelwright 1974, 280–281). In modern times the dried root bark of the bayberry has found value as an astringent and emetic (*Merck index* 1983, 143), and was listed in the *National Formulary* (NF) until 1936. An infusion of the berries of the sweet gale, a species native to America, was said to be a good remedy for itch, or for worms (Millspaugh 1974, 642). American Indians used the bark as a blood purifier, for venereal disease, and for kidney disorders (Moerman 1977, 121).

The wax myrtle family is represented by only one species in Alaska, *Myrica gale*, the sweet gale, which grows in shallow water or swamps. The Tanaina boiled the leaves and drank the tea for tuberculosis, and used it as a wash for pimples and boils. The whole plants were sometimes employed as switches in the steambath (Kari 1981, 30).

Betulaceae (Birch family)

The Betulaceae, which include the birches and alders, have long been known in Europe as a source of several important medications. In particular, distillation of the bark and small twigs of the birch produced sweet-birch oil, a form of methyl salicylate, or oil of wintergreen, which was used as a diuretic, stimulant, and astringent (Vogel 1970, 281). It is still available today as a component of more than a dozen commercial liniments (Bullips and Bullips 1987, 385). Rectified birch-tar oil was listed in the NF until 1955 and used externally as a parasiticide, and for the treatment of chronic skin diseases such as psoriasis (Reynolds and Prasad 1982, 507). Some American Indians used birchbark as a poultice for wounds, cuts, and burns, while others drank an infusion of the inner bark for colds and cough. A few tribes employed birch products for their diuretic effects, as a remedy for stomach cramps, or as a salve for sores or ringworm (Vogel 1970, 280–282).

Alder derivatives were prized for their healing effects in medieval Europe, used externally for wounds and internally as an astringent (Le Strange 1977, 15–16). An alder derivative, called black alder, was in the *USP* until 1894, as a tonic and astringent, and was also used in the treatment of worms, diarrhea, and skin disease (Vogel 1970, 270–271). Two substances isolated from the stem bark, lupeol and betulin, have been shown to have some antineoplastic properties (Viereck 1987, 5). Indians took the tree products internally for constipation, dysentery, cramps, and fever, while they applied them externally for burns, cuts, sores, and hemorrhoids (Vogel 1970, 270–271).

The birch family in Alaska is represented by both genera and both have been used for medicinal purposes. The leaves of *Betula exilis* (presumably *B. nana*, or dwarf birch) were used on Nunivak Island to prepare a tea taken for stomach-ache or intestinal discomfort (Lantis 1959, 5). The Tanaina found the sap of *B. kenaica* (Kenai birch), and perhaps *B. papyrifera* (paper birch) helpful as a spring tonic, and applied it externally to sores and boils (Kari 1981, 13–15). The Atka Aleuts were said to use burnt birchbark as a remedy (Black 1984, 173), although according to Hultén no species of birch lives as far west as Atka. The Ingalik Athapaskans prepared a lotion for pimples by boiling the inner bark fibers of some kind of birch (Osgood 1958, 229). Other Athapaskans boiled the sap and applied it to cuts to prevent infection (Hall 1979).

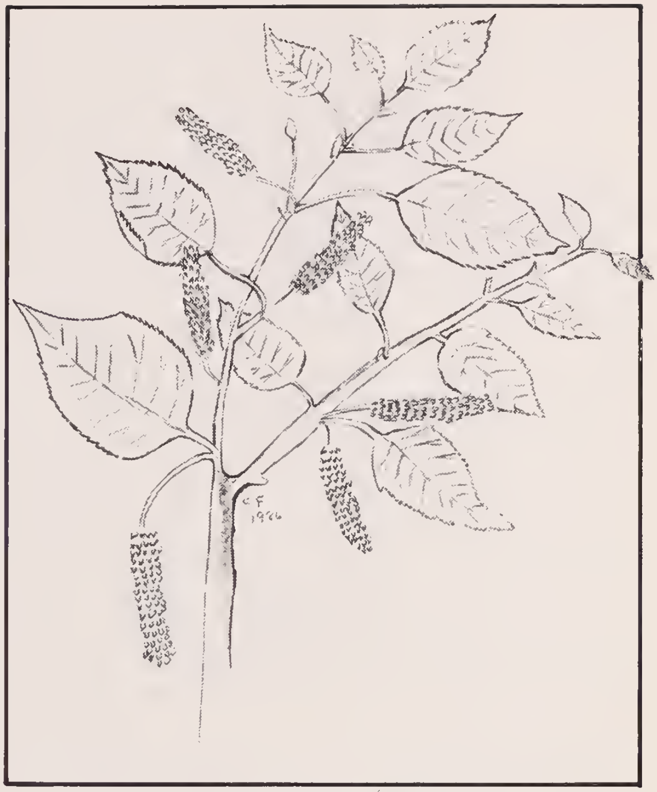

Figure 9 ***Betula papyrifera:*** **paper birch, white birch**

The two main varieties of *Alnus crispa*, the mountain alder in the interior and the Sitka alder in southeastern Alaska, were probably those principally employed for healing. The Tanaina drank a tea, prepared by boiling the inner bark of the alder, for the treatment of tuberculosis, fever, and excessive gas (Townsend 1965, 345–346; Kari 1981, 20, 22). The Chandalar Kutchin, on the other hand, boiled the buds and administered the tea for venereal disease (McKennan 1965, 71). Around Prince William Sound the Chugach Eskimos treated diarrhea by making an infusion from the female cones (Wennekens 1985, 44). (In this regard it is interesting to note that Captain Nathaniel Portlock [1789, 252], sailing near these shores in 1787, found a similar tea to have marked laxative and emetic properties.) The Chugach also made switches from alder branches to flagellate themselves in the steambath for the treatment of arthritis and sore muscles (Wennekens 1985, 44). In southeastern Alaska

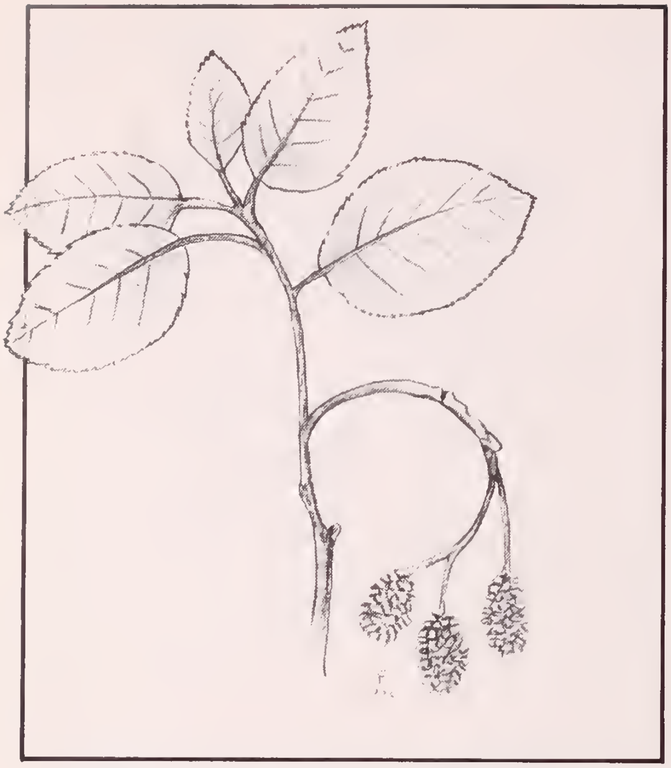

Figure 10. ***Alnus crispa:*** **American green alder, mountain alder**

the Tsimshian drank an infusion of alder bark for stomach ulcers, nosebleeds, and cuts, and applied it externally to open sores (McGregor 1981).

Urticaceae (Nettle family)

Members of the nettle family, characterized by the stinging hairs on the stem, have been used for healing purposes throughout the world. In ancient times and even extending into the Middle Ages patients suffering from paralysis were switched with these prickly plants, a practice known as urtication (Wheelwright 1974, 23). Various extracts have found application as diuretics or astringents, as a soothing ointment for poison ivy, and even as a stimulant for hair growth (Millspaugh 1974, 609; Lewis and Elvin-Lewis 1977, 340). In Europe nettles were prized as an antidote against several plant, insect, and reptile poisons (Le Strange 1977, 251). Some species of *Urtica* have been found to contain the neurotransmitter serotonin (Reynolds and Prasad 1983, 1753). Among Indians the nettle found use for colds, diarrhea, kidney trouble, rheumatism, and paralysis (Moerman 1977, 196–197).

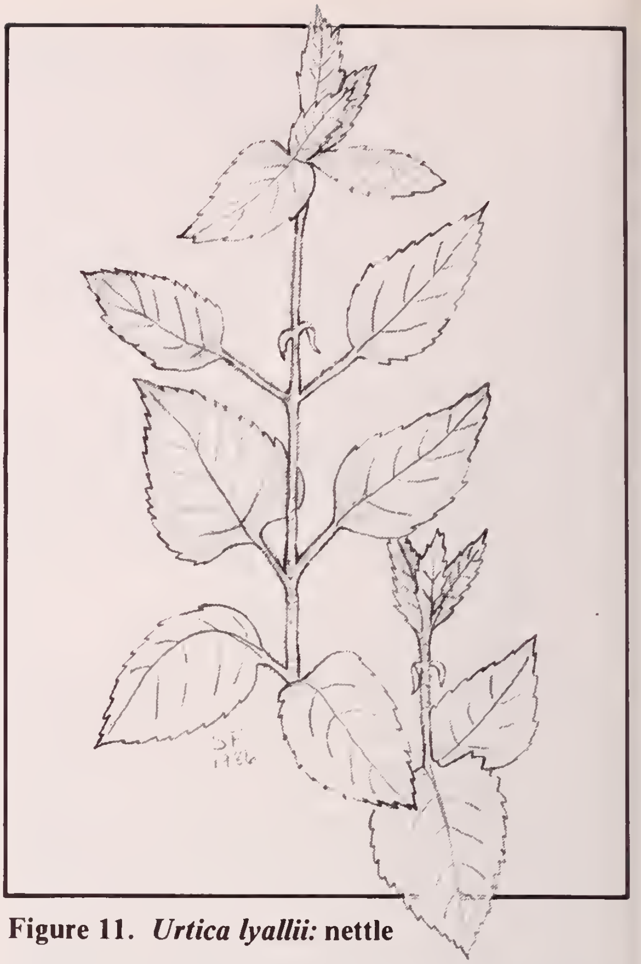

Figure 11. ***Urtica lyallii:*** **nettle**

Three species of nettles occur in Alaska, only one of which, *Urtica lyallii*, has been associated with healing. The people around Prince William Sound used an infusion of nettle roots to treat constipation (Birket-Smith 1953, 117). The root itself was sometimes cleaned and crushed, then heated on a hot rock. An individual suffering from toothache then draped a blanket around himself and the rock, poured some water on the rock, and inhaled the steam. Following this a small piece of nettle root was applied directly to the tooth, which next morning was said to be easily removed. A second toothache treatment involved placing the warmed root directly over the tooth, together with a small mat of woven roots outside the cheek. According to the informant, the offending tooth would crumble and could be easily removed after three or four treatments (Wennekens 1985, 44–45). A Koniag treatment for toothache involved washing and pounding the root and holding it over the painful tooth. On Kodiak Island a tea from nettle roots was also thought to be good for stomach troubles, and one prepared from the leaves was administered as a tuberculosis remedy (Preston 1961; Graham 1985, 49). The Tanaina placed heated nettle leaves directly over arthritic joints (Osgood 1937, 116). The people of Kodiak Island prepared an infusion from the leaves and administered it as a tuberculosis remedy (Graham 1985, 49).

Santalaceae (Sandalwood family)

Geocaulon lividum, sometimes kown as the northern commandra, or timberberry, was the only representative of the sandalwood family used medicinally in Alaska. The Tanaina Indians chewed or mashed the leaves and applied them to skin sores. Internally, they chewed the berries or made a tea from the roots and leaves as a remedy for sore throat, stomach trouble, or tuberculosis (Kari 1981, 70).

Polygonaceae (Buckwheat family)

The Polygonaceae are a family of herbs and shrubs found chiefly in the north temperate climates. Although some members contain toxic substances, several plants have important commercial as well as local value as food.

A species of *Polygonum* was thought to have been used medicinally by Hippocrates, and others (or the same one) have been employed in Europe for centuries as astringents (Wheelwright 1974, 72, 188). The plant is also a popular remedy in China (Viereck 1987, 13). A few species of *Polygonum* have a high tannic acid content, which some have considered carcinogenic. Others contain anthraquinones, which have a strong purgative action (Lewis and Elvin-Lewis 1977, 119, 203–204). Indians used the plant as an anti-diarrheal and hemostatic (Moerman 1977, 144–145).

Members of the genus *Rumex* have long been known as potent antiscorbutics and laxatives. The dried root is cathartic and astringent (*Merck index* 1983, 1194). Most species of *Rumex* contain oxalic acid, which may reach a toxic level in some (Lewis and Elvin-Lewis 1977, 20, 34). Among American Indians *Rumex* species were used to treat many conditions, including diarrhea, colds, sores, boils, jaundice, and rheumatism (Moerman 1977, 166–169). A special use was for the treatment of skin rashes due to *Urtica*, which often grows close association (Lewis and Elvin-Lewis 1977, 347).

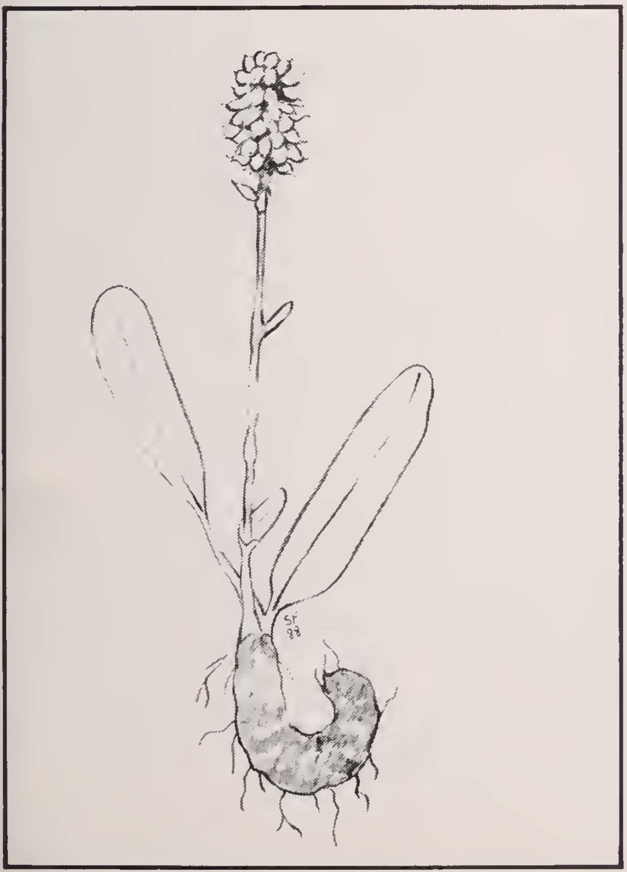

Figure 12. *Polygonum bistorta:* bistort, *makarsha,* pink plumes, wild rhubarb

The buckwheat family is widely distributed in Alaska, several members having significance to the Alaska Natives both as food and as medicines. The best known plants of this group go by the common names sourdock, arctic dock, sourgrass, sorrel, wild spinach, and Indian rhubarb.

In the early nineteenth century Father Ivan Veniaminov (1984, 293) reported that the Aleuts used an astringent root called *makarsha* (probably *Polygonum bistorta*) as a remedy for diarrhea. Another plant of this genus, *Polygonum alaskanum*, or wild rhubarb, was an important food plant as well as having some medicinal virtues. The Ingalik cooked the leaves with fish eggs, strained the mixture, and drank it warm as a laxative (Osgood 1958, 230). Other Athapaskans drank an infusion made from the roots for some type of heart trouble (L. Anderson in Smith 1973), or to purify the blood (Hall 1979).

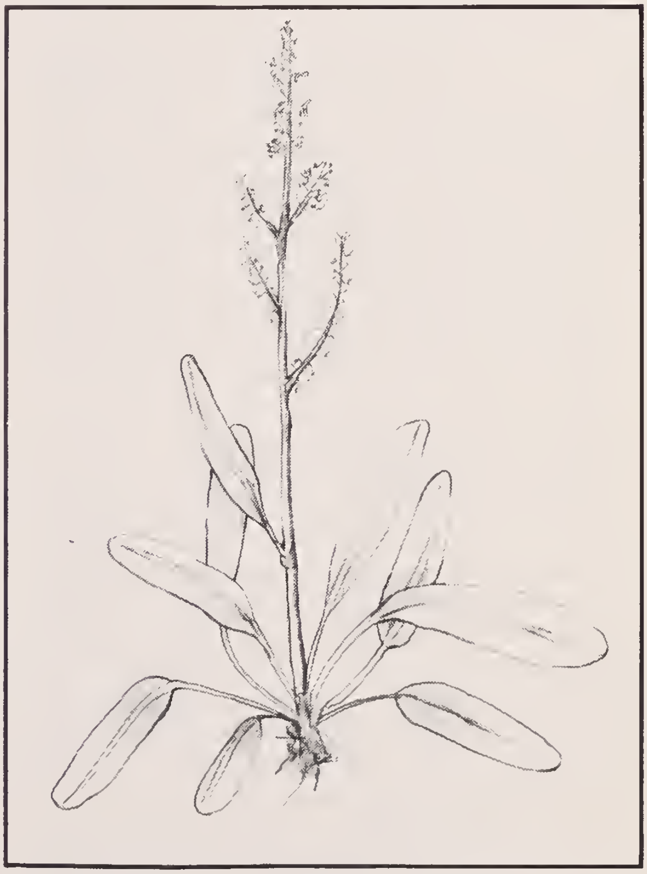

Figure 13. *Rumex arcticus:* arctic dock, sourdock

Rumex acetosella, or sheep sorrel, was apparently introduced to the Pacific rim by the Europeans, but has been adopted by the Aleuts of Atka, who steamed the leaves and applied them to warts and bruises (Hultén 1968, 375; Bank 1953). Among the Chugach *R. fene-*

stratus leaves were dried, pulverized, and taken as snuff to combat sneezing and headache (Wennekens 1985, 45).

The best known species of *Rumex* in Alaska is the widely distributed *R. arcticus*, usually known as sourdock, or arctic dock. The Chugach boiled the roots and employed the tea as a laxative (Wennekens 1985, 45). Their neighbors the Tanaina also knew this use, but in addition took it as an emetic and to treat such diverse conditions as hangover, bladder trouble, stomach trouble and tuberculosis (Kari 1981, 95).

Other sources do not identify the *Rumex* species. A report from the 1790s indicates that the Aleuts used the roots and leaves of sorrel in the treatment of wounds (Merck 1980, 176).

Another account from around this period, from Kodiak Island, described the people using an infusion of crushed sorrel called *atunat'* as an emetic and sedative (Gedeon *in* Pierce 1978, 130; Black 1977, 96). More recent information from Kodiak suggests that raw sourdock leaves were chewed to purge the bowel or cleanse the blood (Graham 1985, 11–12). The Chugach used an infusion from the root as a remedy for constipation (Birket-Smith 1953, 117).

Among other Eskimo groups, the boiled leaves and stems of the same plant, in apparent contradiction, were used as a treatment for diarrhea (Oswalt 1957). On the North Slope the Inupiat ate sourdock cooked or raw for diarrhea, usually mixing it with seal oil. This latter concoction, incidentally, sometimes served also as a remedy for measles (DeLapp and Ward 1981, 31, 42).

The Chandalar Kutchin ate the raw roots as a cure for colds (McKennan 1965, 71). Around Yakutat the Tlingit cooked and mashed the roots, mixed them with seal oil, and applied them to cuts (De Laguna 1972, 656).

Nymphaeaceae (Water lily family)

Members of the water lily family, which includes the lotuses and pond-lilies, are found worldwide. Some species of the genus *Nuphar*, the American pond lily, contain lutenuran, which has antibacterial, antifungal, and anti-trichomonal effects (Reynolds and Prasad 1983, 1724). Indians were said to have eaten a "kind of Nymphaea" to prevent scurvy (Vogel 1970, 249). Some Indians used a decoction prepared from the roots to treat vaginal discharge, or as a poultice for suppurating glands. The plant was also thought to hasten blood clotting in wounds (Millspaugh 1974, 67).

The only common Alaskan species in this family is *Nuphar polysepalum*, or the yellow pond lily, which has a wide distribution. The Chugach burned the whole plant to ashes and applied the powder to open wounds or skin infections (Birket-Smith 1953, 117). An infusion made from the roots was also drunk, but the exact purpose is unknown today (Wennekens 1985, 46). Around Yakutat the Tlingit used roots of the yellow pond lily, although in a somewhat magical context, for sores and tuberculosis, among other things (De Laguna 1972, 658). A more empirical use ascribed to the Tlingit was to make a poultice from the crushed roots and apply it to swellings or bruises (H. W. Alberts *in* Smith 1973).

Ranunculaceae (Crowfoot family)

The crowfoot family of flowering plants is named because many of its members have lobed leaves or leaf formations that resemble a crow's foot. The group includes some of the most familiar wild or cultivated flowers, such as the buttercup, marigold, columbine, anemone, and larkspur. Many contain an acrid juice and some a deadly poison. One member, *Aconitum napellus*, or monkshood, has been known since Roman times as a powerful poison, but was long retained by the *British Pharmacopoeia* mainly as a component of liniments (Wheelwright 1974, 82, 84). The genus *Caltha* also contains aconite, which in excess can cause numbness, weak pulse, convulsions, and respiratory paralysis (Lewis and Elvin-Lewis 1977, 30). A member of the anemone group, hepatica (or liverwort), was used in antiquity for many conditions, including liver disease, and in medieval times for leprosy (Le Strange 1977, 18). Members of the genus *Anemone* and *Caltha* contain the irritant toxin protoanemonin, which has some antibacterial action (Lewis and Elvin-Lewis 1977, 30, 360). Other toxic principles from this family include elatine, which has a paralytic effect similar to tubocurarine (Reynolds and Prasad 1982, 990), and delphinine, causing stomach upsets and nervous symptoms (Lewis and Elvin-Lewis 1977, 30–31), both from the genus *Delphinium*. A substance isolated from *Thalictrum*, thalicarpine, is reputed to have both antibacterial and antineoplastic effects, as well as causing hypotension (Lewis and Elvin-Lewis 1977, 133; *Merck index* 1983, 1324). Much more valuable medicinally was the genus *Ranunculus*, which had wide application among ancient Greek, Arab, and later physicians, who used it primarily externally of its toxicity (Millspaugh 1974, 8–10). The fresh herb of *R. bulbosus* was official in the *USP* until 1882, for use externally as a counterirritant.

Among American Indians various species of *Ranunculus* were used as an astringent, as a snuff to promote sneezing, as a styptic, and as a cold remedy. One interesting application was to crush the leaves in the hands and inhale them to relieve headache (Vogel 1970, 297). *Anemone* species were used to treat colds, rheumatism, tuberculosis, and even insanity. *Thalictrum* extracts served as a love medicine among some Indians, as well as treatment for venereal disease, colds, and fevers (Moerman 1977, 13–14, 190–191).

In Alaska the marsh marigold, *Caltha palustris*, had medicinal uses primarily among the Yupik, although the species itself is widely distributed. Lantis mentions that a mushy cooked preparation of what was probably the marsh marigold was fed to infants with diarrhea, although she also cites the same plant as a treatment for constipation (Lantis 1959, 14–15, 59).

What Blaschke (*in* Krause 1956, 283) calls *Coptis macrocephala* (probably *C. asplenifolia* or *C. trifolia*), or gold thread, was boiled and taken by the Tlingit as a remedy for pneumonia.

The roots of *Delphinium glaucum*, or larkspur, were boiled, washed in water, and the water applied externally by the Tanaina for the treatment of lice or fleas. An infusion from the roots was drunk for tuberculosis, or, alternatively, the boiled roots were applied externally as a poultice (Kari 1981, 117).

Various forms of *Anemone narcissiflorum* are found everywhere in Alaska, but medicinal uses were apparently few. The Unalaska Aleuts boiled the roots until all the juice was extracted and then administered the latter for hemorrhage (Bank 1953). Far away on the upper Yukon, the Chandalar Kutchin macerated and boiled the leaves and applied them as an astringent poultice to cuts and wounds (McKennan 1965, 71).

Thalictrum sparsiflorum, or meadow rue, had some use among the people of Prince William Sound, who prepared an infusion for pneumonia by boiling the leaves, and for tuberculosis by boiling the roots (Wennekens 1985, 46).

Crassulaceae (Stonecrop family)

The stonecrop family of fleshy herbs is distributed worldwide, especially in dry regions. Some types were used by Anglo-Saxon physicians in medieval times (Wheelwright 1974, 105). In a more recent era, stonecrop derivatives have been recommended both in Europe and America as an internal medicine for dysentery, cancer, and epilepsy, and as an external preparation for inflamed mucous membranes, such as those of the throat and vagina (Millspaugh 1974, 224, 226).

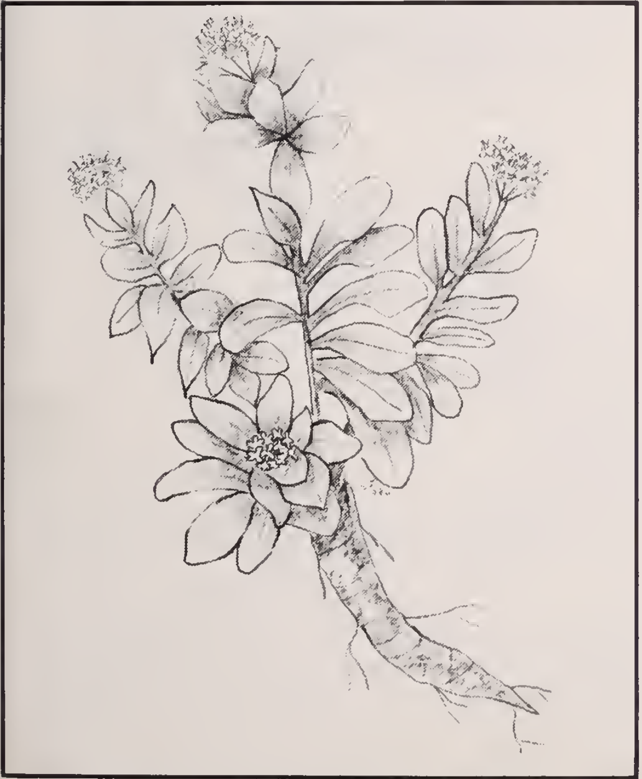

Figure 14. ***Sedum rosea:*** **roseroot, king's crown, stonecrop**

In Alaska there are two genera and three species of stonecrop, only one of which, *Sedum rosea*, or roseroot, has a wide distribution. Usually found in rocky areas, the plant has served both as a food and as a medicine, the latter by the Tanaina and the Yupik Eskimos. On Nelson Island in southwestern Alaska the people chewed the raw roots (but spit out the juice) as a treatment for sore mouth (Ager and Ager 1980). Both there and on Nunivak Island the flowers were eaten as a treatment for tuberculosis. When boiled the flowers also served as a remedy for stomach-ache on Nunivak Island (Lantis 1959, 5, 24, 60). In the Cook Inlet region the Indians prepared an infusion from the leaves and administered it for colds, sore throat, and sores in the mouth. The roots were softened by chewing and then placed on cuts to facilitate healing. A tea from the roots and leaves made a soothing eyewash for sore eyes (Kari 1981, 129).

Saxifragaceae (Saxifrage family)

The saxifrages are a large family of plants found pre-eminently in the colder regions of the world. A preparation called "Black currant," made from the genus *Ribes*, is still in the *British Pharmacopoeia* because of its high ascorbic acid content (Reynolds and Prasad 1982, 1657). American Indians used *Ribes* species for sore throat, tuberculosis, venereal disease, gynecological problems, sore eyes, and skin sores (Moerman 1977, 161–163).

A few species found medical application in Alaska. In the Aleutians the Atkans prepared an infusion from the leaves of the leather-leaved saxifrage, or *Leptarrhena pyrolifolia*, which they gave to persons with influenza (Bank 1980).

In the Yakutat region the berry of *Ribes bracteosum*, called the stink currant (or sometimes the blue currant), was thought to be helpful for syphilis. Likewise, an infusion from the leaves and stems of the fetid, or skunk currant (*R. glandulosum*), was a popular remedy for tuberculosis (De Laguna 1972, 657). The Chugach cut and peeled the branches of the northern black currant, *R. laxiflorum*, and boiled the cambium. After letting the fluid settle, they applied the fluid to sore eyes and cataracts (Wennekens 1985, 47). The Tanaina prepared a similar mixture, using the tea internally as a remedy for colds, influenza, and tuberculosis, and externally as an eyewash (Kari 1981, 52). Another source described skinning and boiling the berry and using the mixture as a poultice for sore eyes (Osgood 1937, 116). Finally, the Eyak Indians of the Copper River Delta boiled the leaves of an unspecified type of currant and applied the extract to sore eyes (Birket-Smith and De Laguna 1938, 218).

Rosaceae (Rose family)

The Rosaceae, a large and varied family of shrubs and trees, include not only roses, but many types of edible berries, most common fruit trees, and familiar flowers such as goatsbeard, cinquefoil, brunet, and avens. Members of the rose family were widely employed as medicines in ancient times. In the Middle Ages and Renaissance rose products often made up one or more components of various ointments and other complex prescriptions (Wheelwright 1974, 116, 153). Today rose products are still widely used in medicine. Red-rose petal, as an infusion, has mildly astringent properties, and rose oil can be found in lozenges, dentifrices, and ointments (Reynolds and Prasad 1982, 429, 682). Rosehips are still found in the USP and are widely taken for their vitamin C content (Billups and Billups 1987, 538). *Geum rivale*, or water avens, was listed in the *USP* until 1882 as an anticoagulant and antipyretic, and was often prescribed for dysentery, colic, debility, dyspepsia, and leukorrhea. Extracts of the wild strawberry, *Fragaria vesca*, were popular remedies for gout, dysuria, gonorrhea, and diarrhea. Blackberry bark, from *Rubus spectabilis*, is still available as an antidiarrheal (*Merck index* 1983, 1194). The mountain ash (*Sorbus* spp.), particularly the bark, also found many proponents and was sometimes administered for malaria in preference to cinchona bark, or for scurvy (Millspaugh 1974, 213, 217, 220–221). The leaves and flowers of some species of *Spiraea* are said to have an antibacterial action (Lewis and Elvin-Lewis 1977, 362).

Indians of the New World had several uses for *Geum* species, especially for intermittent (probably malarial) fevers. Others took extracts for soreness of the chest, cough, rheumatism, smallpox, and for female disorders (Vogel 1970, 388; Moerman 1977, 81–82). Some tribes employed *Fragaria* species for stomach trouble, diarrhea, menorrhagia, and colic (Moerman 1977, 77–78; Viereck 1987, 69). *Sorbus* had relatively little use except by some groups for gastrointestinal upsets, constipation, and colds. Members of the genus *Rubus*, on the other hand, were very popular Indian remedies, especially for diarrhea, hemoptysis, and eye problems. Extracts of the genus *Rosa* were employed as a tonic or stimulant, and to treat, among other conditions, stomach troubles, diarrhea, colds, burns, and itching (Moerman 1977, 163–166, 184).

The rose family is very well represented in Alaska, making up nearly one third of the woody plants native to the state. It includes not only typical flowering plants, but also many types of edible berries.

Dr. Blaschke (*in* Krause 1956, 284) described the use of *Spiraea aruncus* root (now called *Aruncus sylvester*) as an infusion for blood disease. Species of *Aruncus*, or goatsbeard, were used by the Yakutat Tlingit for the treatment of rheumatism, tuberculosis, and perhaps smallpox. The roots were boiled fresh in the spring, and the infusion, when administered, was said to make the patient initially feel worse (De Laguna 1972, 657).

Another early remedy mentioned by Blaschke (*in* Krause 1956, 283–284), used for pleurisy, was a tea made from a type of crabapple that he calls *Pyrus sambucifolius*, but is now called *Sorbus sambucifolia*. As a tuberculosis remedy the Tlingit prepared an infusion from the roots of a second kind of mountain ash, *S. sitchensis* (De Laguna 1972, 657). The Tanaina Indians around Lake Iliamna ate the berries from the tops of the mountain ash for tuberculosis. They also made an infusion from the berries as a cough medicine, or chewed the inner bark for constipation (Townsend 1965, 346). The inner bark and fresh berries were in fact used for several types of illness, including sore throat, tuberculosis, sores, and dysuria. Sometimes the berries were boiled and soaked before they were eaten. Finally, the mountain ash made good switches for use in the steambath. (Kari 1981, 64).

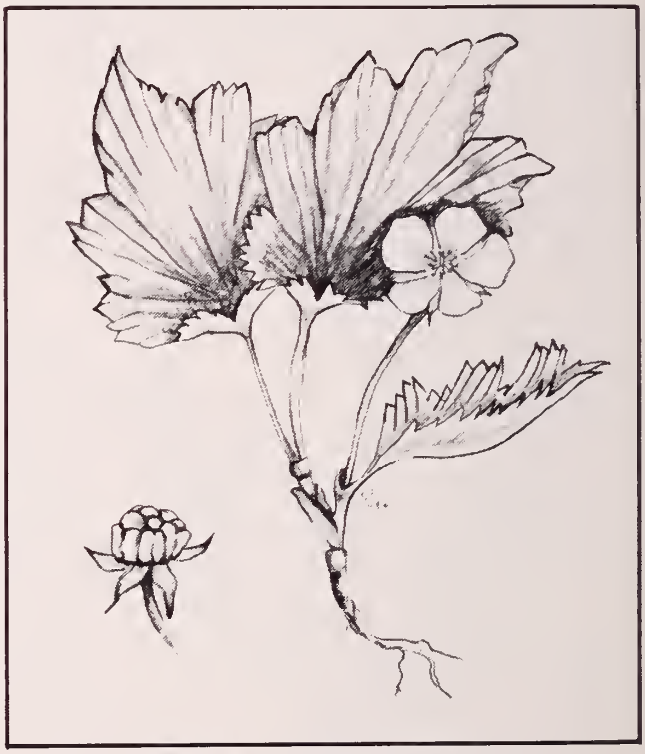

Figure 15. *Rubus chamaemorus:* cloudberry, "salmonberry"

The Koniag placed green or dried salmonberry leaves (*Rubus spectabilis*) over an unhealed wound or infection (Graham 1985, 115–116). On the lower Kuskokwim some Yupik Eskimos ate raw "salmonberries" (probably cloudberries, or *R. chamaemorus*) to treat diarrhea (Lantis 1959, 15). Around Yakutat the Indians drank an infusion of the leaves and stem of the thimbleberry (*R.*

parviflorus) as a tuberculosis remedy (De Laguna 1972, 657).

An infusion prepared by boiling the whole tundra rose, or more correctly the shrubby cinquefoil (*Potentilla fruticosa*), was sometimes administered by the Chugach for gas pains (Wennekens 1985, 47).

The Tanaina had several medicinal uses for members of the genus *Rosa*, or wild rose. They soaked the flowers in hot water, then applied the cool infusion as an eyewash. Another remedy was made by boiling the stems and branches and drinking the tea for colds, fever, stomach trouble, and difficult menses. A tea made from the bark was used as an emetic (Kari 1981, 60).

Finally, in the rose family, the Aleuts employed *Geum calthifolium*, a type of avens. For sores and wounds they prepared a poultice by boiling the leaves, while for colds and sore throats they made an infusion from the roots (Bank 1980).

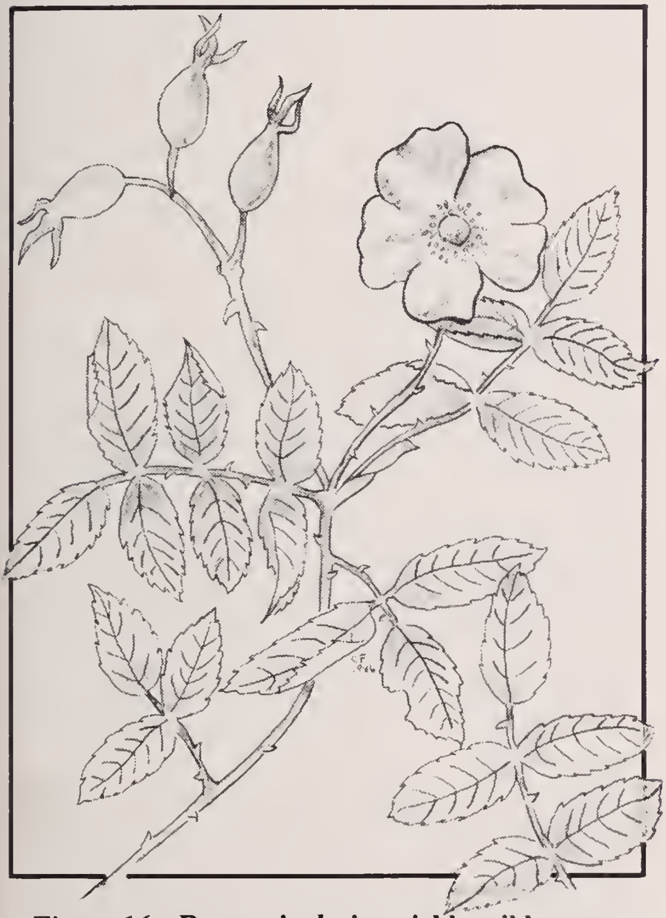

Figure 16. ***Rosa acicularis:*** **prickly wild rose**

Leguminosae (Pea family)

The huge pea family of herbs, shrubs, and trees is distributed worldwide and serves as an important food source. Also known as pulses, most of the plants are characterized by seed-carrying pods. Among those native to America are literally dozens that have found medicinal application, most in topical preparations (Millspaugh 1974, 179–199). A substance isolated from the genus *Oxytropis* may have teratogenic properties (Lewis and Elvin-Lewis 1977, 93). It is of interest that some members of this genus, although perhaps not those in Alaska, contain a poisonous substance called locoine, which causes animals to stagger, lose muscular control, and sometimes die (Heller 1963). Use among the American Indians was limited.

Although nine genera of the pea family are represented in Alaska, only two species are known to be used medicinally. An extract of the root of an *Oxytropis* species was employed by the Tlingit in earlier times as a treatment for colic (Blaschke *in* Krause 1956, 284). On the North Slope, eating so-called "Eskimo potatoes" (*Hedysarum alpinum*) was considered a useful remedy for pinworms (DeLapp and Ward 1981, 66).

Geraniaceae (Geranium family)

The geranium family consists of herbs and small shrubs, both wild and cultivated. Species of this group have been described in ancient herbal books (Wheelwright 1974, 107). In early America the wild geranium (*Geranium maculatum*) was recommended for fevers, canker sores, and dysentery. Geranium root was in the *USP* until 1916 and in the *NF* until 1936, for use as a tonic and astringent. An extract of the plant has been found to lower the blood sugar. In Bulgaria *Geranium* has been used as an aphrodisiac (Lewis and Elvin-Lewis 1977, 219, 329).

Some North American Indians treated venereal disease, probably gonorrhea, with geranium, and also administered it for thrush and hemoptysis. Externally it was considered a good styptic and a remedy for vaginal discharge, sore mouth, and toothache (Vogel 1970, 390–391).

In Alaska the geranium family is represented by only three species, one of which, *Geranium erianthum*, was widespread and used medicinally. On Atka an infusion from the leaves was taken as a gargle for sore throat (Bank 1980). Around Cook Inlet the Indians chewed the raw roots for mouth sores, and made a root infusion that was drunk for various illnesses, including sore throat, mouth sores, ulcers, tuberculosis, diarrhea, heart trouble, and to "clean out the toxins" from new mothers (Kari 1981, 120). The Chugach used the plant in some manner not now clear to stop bleeding (Wennekens 1985, 47–48). On Kodiak Island geranium roots were chewed as a tuberculosis remedy (Preston 1961).

Elaeagnaceae (Oleaster family)

The oleasters are a family of shrubs with leathery leaves and a dense covering of hairs. They are found principally in rocky areas of the Northern Hemisphere. American Indians have used extracts for cough, stomach troubles, constipation, and gonorrhea (Moerman 1977, 177).

Only two plants of this family have been described in

Alaska, one of which, *Shepherdia canadensis*, or soapberry, was used for healing. The fruit contains a bitter substance called saponine (Hultén 1968, 684). The Tanaina boiled the stems and drank the infusion for tuberculosis, or used the liquid for washing cuts or soaking swollen areas. (Kari 1981, 34)

Onagraceae (Evening primrose family)

The evening primrose family has a worldwide distribution, most of its members growing as herbs in the temperate and northern parts of North America. The genus *Epilobium*, or willow herb, has been used by some as a tonic or astringent, useful in mild diarrhea or cramps. The evening primrose itself (genus *Oenothera*) has been considered helpful externally for infant rashes, and internally for asthma, pertussis, irritable bladder, and severe diarrhea (Millspaugh 1974, 231–235). Some Indians used the plant to make poultices for boils and burns (Moerman 1977, 68).

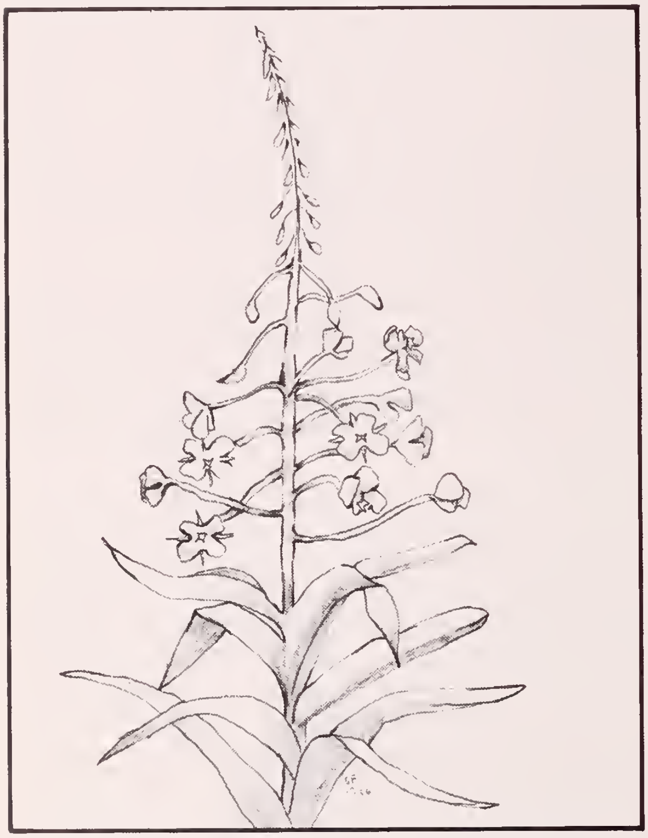

Figure 17. *Epilobium angustifolium:* tall fireweed, willow herb

Of the two genera and 15 species occurring in Alaska, only one species, *Epilobium angustifolium*, or tall fireweed, is recorded as having medicinal use. The Chugach prepared an infusion from the roots as a laxative. Another infusion, possibly made from the leaves, was used to stimulate milk flow in a woman after childbirth (Birket-Smith 1953, 117). In a more recent survey, the people of Prince William Sound cleaned and crushed the root, placed it on small cuts and boils, and bandaged over it (Wennekens 1985, 48). The Tanaina used the raw stems for similar purposes (Kari 1981, 116). In southwestern Alaska a tea was prepared from the leaves that the people of Nelson Island used for constipation, and the people of Nunivak Island for stomach-ache (Ager and Ager 1980; Lantis 1959, 5).

Araliaceae (Ginseng family)

The *Araliaceae* include herbs, shrubs, and trees, mostly prickly, which are found around the world but particularly in tropical climates. The best-known representative is undoubtedly the true ginseng itself (*Panax schinseng*), which was a favorite Chinese remedy for nearly every conceivable illness. In fact, the plant was so highly esteemed in China that the emperor himself had a monopoly on who could gather the roots (Millspaugh 1974, 277). A closely related species, *P. quinquefolia*, was once common in North America and sold to China in such quantities that it has all but become extinct. The plant remained in the *USP* until 1882 as a stimulant and stomachic (Vogel 1970, 307–310). A derivative of *Oplopanax japonicum* is used commercially today in Japan as an antipyretic and antitussive. Chemical analysis of members of this family have demonstrated that most contain a substance causing hypoglycemia, a property which has been exploited by some native peoples for the treatment of diabetes (Turner 1982).

Since Indians were often employed by early European settlers to gather ginseng for the commercial market, it is possible that their own use of the plant was influenced by the enthusiasm shown for it by the Whites. Whatever the truth of this assertion, the native peoples of America found many medicinal uses for the plant themselves. Some took it to control bleeding, as a fertility drug, or for headache, fever, female troubles, shortness of breath, and cough. Others considered an infusion of the root helpful for vomiting, rheumatism, and as a general tonic (Vogel 1970, 307–310). Among the Indians to the south and east of Alaska, Nancy Turner (1982) has collected many examples of how devil's club has been used for healing.

Devil's club, variously called *Echinopanax horridum*, *Oplopanax horridus*, and *Fatsia horrida*, is the single Alaskan representative of this family. Among all the Native groups, devil's club was preeminent—almost a panacea—among the Indians of southeastern Alaska, but it was also used by the Chugach, Eyak Indians, Tanaina, and probably the Koniag—in fact wherever it was found.

The first mention of the medicinal use in Alaska of what may have been this plant was by Gavril Davydov (1977, 177) in the early nineteenth century. He noted that a certain Koniag woman prepared an infusion from the roots of a "sarsaparilla-like" plant which she used to treat venereal disease. The American sarsaparilla plant,

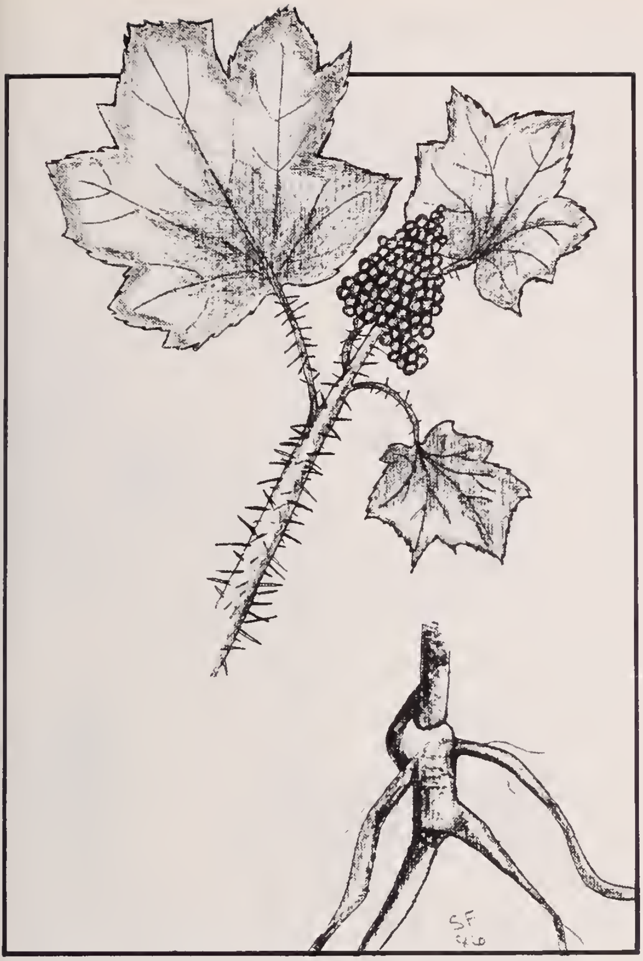

Figure 18. ***Echinopanax horridum:*** **devil's club**

well-known for its healing properties, is a member of the same family as the devil's club, but does not occur in Alaska.

The Tanaina made an infusion from the inner bark of the devil's club for use in colds, cough, tuberculosis, and stomach trouble, while an infusion from the stems and branches they prescribed for fever. They also baked the inner root and applied the powder under a bandage for swollen glands, boils, and sores (Kari 1981, 62).

The Chugach sometimes treated dropsy and other diseases in earlier times by burning the bark to a powdery ash. The patient was stood up against a wall, his ankle tied against his buttock, and an incision made at the heel, onto which the ashes were sprinkled. The victim had to maintain this position until the following morning. Ashes from burned devil's club leaves were also sometimes mixed with salmonberries and dog feces and applied to burns (Birket-Smith 1953, 117), or the ashes simply dusted on the burn and bandaged over. Sometimes the roots were cleaned, crushed, soaked in hot water and applied externally as a poultice for an infected wound or a painful joint. Arthritis was also treated by boiling sections of the unpeeled stems in a tub and bathing in the water. A final external use by the Chugach was to apply the root epidermis to boils. Internally, they made an infusion from the roots for use as an emetic, such as in cases of food poisoning. They also stripped off the cambium layer and boiled it to make a strong tea thought to be useful for arthritis, colds, and pneumonia (Wennekens 1985, 48–49).

The Eyak Indians, neighbors of the Chugach Eskimos, also found the devil's club a useful emetic as well as purgative. For external use, they had the mother and father bathe ten days after childbirth in water prepared by boiling the skin of the plant (Birket-Smith and De Laguna 1938, 160, 204).

The Tlingit had multiple uses for different parts of the plant. The inner bark, in particular, was thought to have healing powers. An infusion could be drunk for colds, pneumonia, or arthritis (McGregor 1981), or mixed with seal oil and taken as an emetic or cathartic (Alberts *in* Smith 1973). Chewing the bark also produced vomiting and diarrhea (De Laguna 1972, 659). An infusion made from the bark or leaves could be taken to treat a cold, chest pain, arthritis, black eye, constipation, gallstones, stomach ulcers, or tuberculosis (Justice 1966). A woman with amenorrhea drank a small amount of the infusion, or else squatted over the bark heated on rocks and doused with water to make steam (De Laguna 1972, 659).

The plant likewise had many external uses in Tlingit healing. The inner bark was sometimes dried, powdered, mixed with cedar pitch, and applied to abrasions (Justice 1966). Alternatively, the green inner bark was softened by chewing and then laid on wounds as a poultice (Alberts *in* Smith 1973). Around Yakutat it was reported that a poultice of roasted bark shavings mixed with spruce pitch served admirably for impetigo or "any place that's sore." Shavings were also added to a hot bath as a treatment for pneumonia (De Laguna 1972, 659). Finally, bark strips were laid along a fracture site, inner side to the skin, to relieve pain and swelling (Justice 1966).

The Haida, at least those from the Queen Charlotte Islands, also found devil's club helpful for many kinds of illness. They decocted the inner bark in sea water and drank it over a nine-day period as a laxative and as a treatment for arthritis, and drank an infusion prepared in plain water for tuberculosis of the bone. They chewed the bark and swallowed the juice for colds or generalized illness, and rubbed the berries on the hair and scalp of children to combat lice and dandruff. In the steambath they used the stems as a switch for arthritic joints (Turner 1982).

The Tsimshian Indians were also aware of the laxative effect of the plant, caused either by drinking an infusion or by chewing the stalk (Garfield and Wingert n.d., 40). Some thought the plant useful for improving vision, or even as a treatment for blindness. For insanity the patient's shaved head was beaten gently but repeatedly with the plant until blood was drawn. For venereal disease a preparation of devil's club was taken internally. In recent years it has also been advocated for cancer (McGregor 1981).

It should be mentioned in passing that devil's club had many magical uses in all the cultures of southeast Alaska; for example, in the training of a shaman, for protection against sorcery, and for the guarantee of a successful hunt (Turner 1982; Justice 1966; De Laguna 1972, 660; Krause 1956, 188).

Umbelliferae (Parsley family)

Members of the parsley family, sometimes also called the carrot family, grow principally in the north temperate regions, and are characterized by an umbel, or a number of small flower stalks spreading from a common center. The family includes several important food plants, such as carrots, celery, parsnips, and a number of common garden seasonings. Some plants are poisonous; in fact, the "hemlock" which Socrates was said to have drunk was derived from a member of this family, *Conium maculatum*. The poisonous principle, an alkaloid known as coniine, also has had several medical uses and for many years was listed in the *USP*. A European species of *Angelica* was esteemed as a plague remedy as late as the seventeenth century (Le Strange 1977, 24). An American member of the genus, great angelica (*Angelica atropurpurea*), has also been given for generalized edema, colic, and amenorrhea (Millspaugh 1974, 247–250). *Angelica* species contain coumarins, which have diaphoretic, diuretic, and antiflatulence properties, and a volatile oil which is useful as an expectorant (*Merck index* 1983, 94; Reynolds and Prasad 1982, 687). Members of the genus *Heracleum* also contain coumarins, and have been used both as an anticonvulsant and as an aphrodisiac (Lewis and Elvin-Lewis 1977, 167, 328).

The American Indians may have occasionally used water hemlock (*Cicuta maculata*) for suicide (Millspaugh 1974, 261–265). Some tribes took *Angelica* extracts for coughs, colds, diarrhea, constipation, and intestinal worms (Moerman 1977, 15–16). Siberian natives burned the root and inhaled the fumes for seasickness (Hultén 1968, 705). *Heracleum lanatum* was the source of many Indian skin remedies (Kuhnlein and Turner 1986).

In Alaska 14 genera and 21 species of the parsley family are known. A report from the beginning of the 19th century described boiling parsley (*chinkak*) stalks and roots and mixing them with fat as a treatment for boils (Gedeon *in* Pierce 1978, 130; Black 1977). Among the Tlingit Dr. Blaschke (*in* Krause 1956, 283) identified a type of sweet cicely ("*Osmorhyza brevistyla*"), used in the form of an infusion to treat cough. The people of Atka prepared an infusion from the hemlock parsley, *Conioselinum gmelinii* (now *C. chinense*), for use in sore throat (Bank 1953).

Various species of *Angelica*, known as wild celery or wild parsnip, were a significant food source for the Alaska Natives, but also had several medicinal uses. The earliest report is from the late 18th century, when Dr. Merck (1980, 176) on the Billings expedition described the Aleuts boiling the plant and using it both internally and externally to treat wounds. The Aleut shamans of Atka were also said to use the plant internally for sickness (Black 1984, 173). Other Aleuts applied the leaves of *A. lucida* as a poultice, or sliced the root in two, heated the halves, and placed them over a painful site, even if the pain was internal. The plant was also used in the treatment of colds and sore throats (Bank 1953). Among the Tanaina the roots of *A. lucida* were heated and applied directly to a painful tooth, a procedure which after a time was said to cause the tooth to break up and fall out. The root was also cut up, mashed, boiled, and applied as a poultice to aches, pains, sores, and cuts (Kari 1981, 86). The Eskimos of St. Lawrence Island had great confidence in the medicinal value of *A. lucida*, taking it as a general tonic and as a treatment for most internal illnesses (Young and Hall 1969).

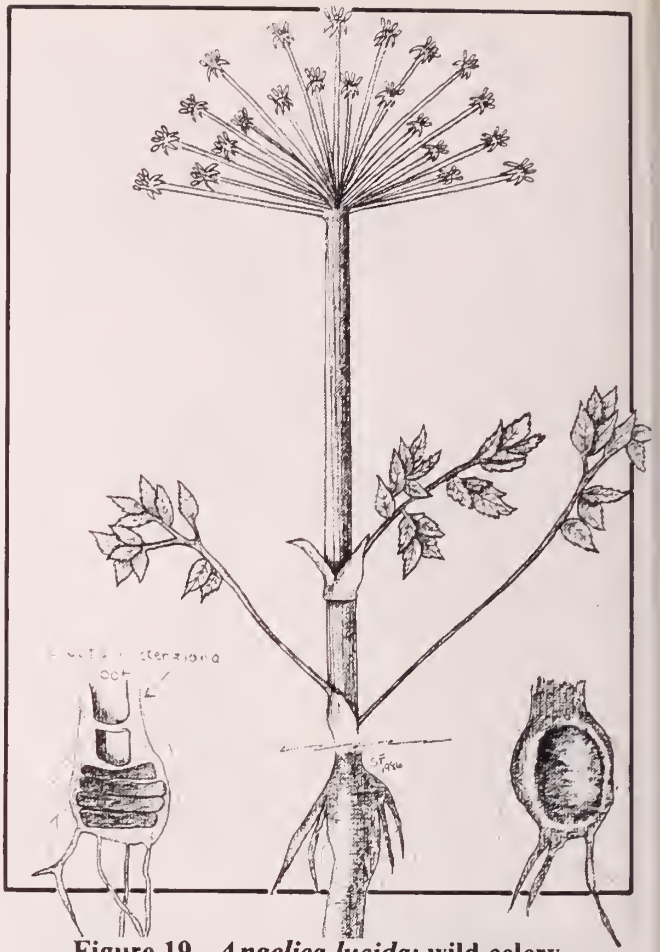

Figure 19. ***Angelica lucida:*** **wild celery, wild parsnip; Also showing root of** ***Cicuta mackenziana:*** **the similar but poisonous water hemlock**

Heracleum lanatum has several common names, in-

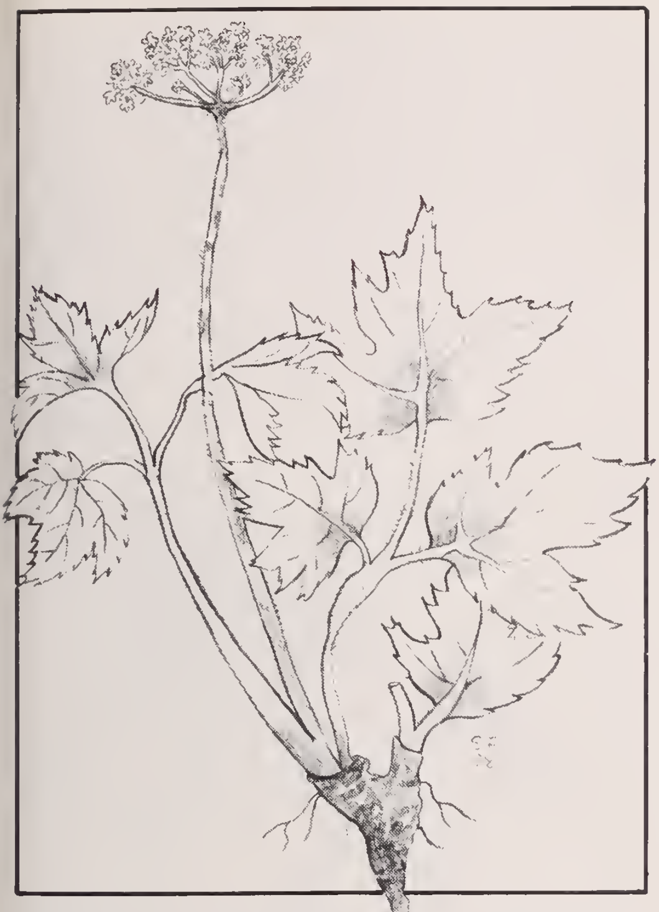

Figure 20. ***Heracleum lanatum:*** **cow parsnip, Indian rhubarb, wild celery,** ***putshke***

cluding Indian rhubarb, wild celery, cow parsnip, and the Russian word *putshke*. The Aleuts prepared a tonic for the treatment and prevention of sore throats (Bank 1953). Around Cook Inlet the Tanaina chewed the roots raw or made an infusion from them for sore throat, mouth sores, or tuberculosis. They also applied the hot roots directly to an aching tooth, or made a poultice from them for cuts, body aches, or arthritis (Kari 1981, 83-84). The Tlingit from around Yakutat also found the steamed and split roots helpful for arthritis (De Laguna 1972, 656). Around Prince William Sound the people added the outer skin to seal oil and after a week applied it to the scalp for dandruff or baldness (Wennekens 1985, 50).

Cornaceae (Dogwood family)

The dogwoods are shrubs or trees of the large genus *Cornus* which characteristically have a small flower surrounded by large petal-like bracts. Among the species to which medicinal value was attributed in the New World were three listed in the *USP* in the nineteenth century: *C. florida* (flowering dogwood), *C. circinata* (round-leaved dogwood), and *C. sericea* (swamp dogwood). The bark of each was used as an astringent or bitter tonic (Vogel 1970, 301). *C. florida* was widely prescribed for malaria, although it was considered less effective than cinchona bark (quinine) (Millspaugh 1974, 282). *Cornus* species contain a tannin that has some antineoplastic effects (Lewis and Elvin-Lewis 1977, 135).

The American Indians found in the dogwood much of value for healing, using extracts for fevers, dysentery, hemorrhoids, as an eyewash, and as a poultice for sores (Vogel 1970, 300-301).

This family has a few members in Alaska, only one of which, *Cornus canadensis* (red bunchberry, creeping dogwood, or dwarf cornel) has been reported to have medicinal value. The Chugach applied the leaves to a wound and bandaged over them (Wennekens 1985, 51). The Ingalik Athapaskans boiled the berries and drank the warm liquid as a remedy for stomach-ache (Osgood 1958, 230). Among the Yakutat Tlingit the leaves were heated on the stove and applied externally for infected cuts, burns, cataracts, and even to the breast of mothers for sore nipples or to stimulate milk flow (De Laguna 1972, 656).

Pyrolaceae (Wintergreen family)

The Pyrolaceae include several North American species that had medicinal value. Some settlers made a tonic and an antiscorbutic from the genus *Pyrola*. The Indians employed extracts for such diverse conditions as fevers, kidney problems, scrofula, arthritis, dysentery, amenorrhea, sore mouth, toothache, and skin disorders (Vogel 1970, 394).

In Alaska one species, *Moneses uniflora* (wax plant, single delight, shy maiden, Star of Bethlehem), is known for its healing properties. The Chugach prepared an infusion from the whole plant for sore throat (Wennekens 1985, 50), whereas the Tlingit used it for coughs and colds (Alberts *in* Smith 1973).

Empetraceae (Crowberry family)

The crowberry family is represented in Alaska by a single species, *Empetrum nigrum*, variously known as the crowberry, blackberry, or mossberry. The Tanaina soaked and boiled the leaf and stem and used the tea, or simply the cooked berries, as a remedy for diarrhea. Crowberry juice was thought to be helpful for kidney trouble. Also around Cook Inlet the people prepared a tea from the root for use as an eyewash (Kari 1981, 47–48). The Eskimos of the North Slope and some Athapaskans applied the juice to the eyes as a treatment for snowblindness (DeLapp and Ward 1981, 18; Hall 1979). In the Kotzebue Sound area, crowberry juice was squeezed into the eye for cataract and among other Native groups the bark of the stem is said to have been used for the same purpose (Mauneluk Cultural Heritage Program 1976, 30; Graham 1985, 90).

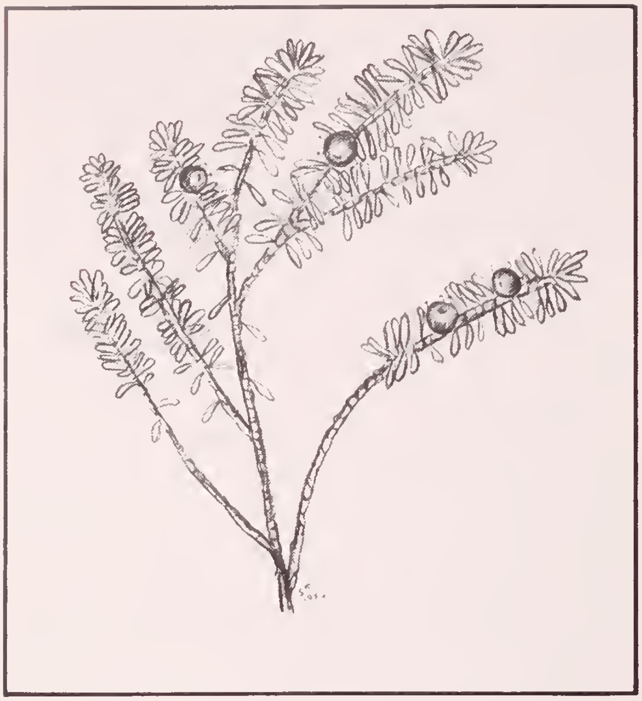

Figure 21. ***Empetrum nigrum:*** **crowberry, mossberry, blackberry**

Ericaceae (Heath family)

Plants of the heath family are for the most part low shrubs which form the cover vegetation of many regions of the world which have wet, acid soils, including moors, swamps, and tundra. The group comprises many common flowers, such as rhododendron, azalea, and mountain laurel, as well as several important edible berries, including blueberries, huckleberries, and lowbush cranberries. The bearberry, *Arctostaphylos uva = ursi*, was employed in medieval times as an astringent and hemostatic, and later as a diuretic and oxytocic. The dried leaves were listed in the USP until 1936. *Arctostaphylos* species contain a glycoside called arbutin, which has diuretic and astringent effects. Crude extracts of the leaves show some antineoplastic activity (Lewis and Elvin-Lewis 1977, 135). Members of the genus *Vaccinium* have been employed as diuretics and astringents, and in the treatment of degenerative retinal disease (Reynolds and Prasad 1982, 1730). The genus *Gaultheria* is the main source of methyl salicylate, or oil of wintergreen, the same active principle that is found in birch oil. Extracts of the mountain laurel *Kalmia latifolia*, have found some application for skin diseases and for diarrhea (Millspaugh 1974, 393, 405). The plant contains a deadly poison called andromedotoxin that causes slow pulse, hypotension, loss of coordination, and progressive paralysis (Lewis and Elvin-Lewis 1977, 35). The leaves are the source of toxic diterpinoids called grayanotoxins, which can also cause hypotension (Merck index 1983, 652–653). *Ledum* species contain ledol, a toxic substance that can cause cramps and paralysis (Viereck 1987, 43–44).

The Indians found bearberries helpful for a few conditions. Some mixed them with tobacco and others used the leaves as an ingredient of remedies for female troubles. Wintergreen berries were sometimes chewed as a stomach tonic, or for rheumatism, fevers, and lumbago. An infusion from the leaves of the genus *Ledum*, or Labrador tea, was popular among the northern Indians as a stimulant and diuretic, and as a treatment for gastrointestinal disorders (Vogel 1970, 169, 280, 394).

The heath family is widely distributed in Alaska, with 13 genera and 30 species of shrubs, many of which bear edible berries.

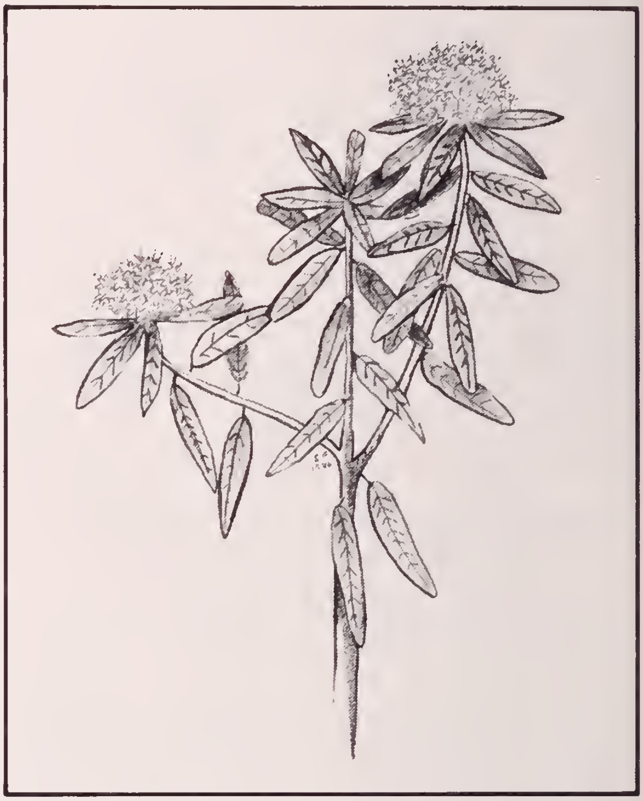

Figure 22. ***Ledum palustre:*** **Labrador tea, Hudson's Bay tea, muskeg tea, Indian tea**

Ledum palustre, sometimes known as *L. decumbens* or *L. groenlandicus*, is the well-known Labrador or Hudson's Bay tea. It had medicinal uses throughout Alaska. The earliest reference is that of Blaschke (*in* Krause 1956, 284), who noted than an infusion made from a mixture of *Ledum*, pine, and cedar was considered by the Tlingit to be helpful for syphilis. Other Indians of the region used it for high blood pressure, to purify the blood, or as a cold or stomach remedy (De Laguna 1972, 657; McGregor 1981). Among Athapaskans the Tanaina prepared an infusion of the leaves they found helpful for many ailments, including arthritis, dizziness, stomach trouble, heartburn, constipation, and even hangover (Kari 1981, 99).

The people of Kodiak Island drank Labrador tea for chest ailments, including tuberculosis (Graham 1985, 38). The Yupik used it for hemoptysis (presumably from tuberculosis) and also to settle an upset stomach (Lantis 1959, 5; Ager and Ager 1980). The leaves were sometimes burned as a fumigant (Lantis 1959, 14). For the Inupiat Labrador tea was used to treat chronic cough or the vomiting resulting from food poisoning (Mauneluk Cultural Heritage Program 1976, 51).

The bearberry, also known as meal berry or kinnikinnik (*Arctostaphylos uva = ursi*), was a Tsimshian remedy for malaise and ulcers. It was also eaten by the Tlingit to treat arthritis, and by the Tanaina as a laxative (McGregor 1981; Kari 1981, 43).

The genus *Vaccinium* in Alaska includes the blueberries, huckleberries, and lingonberries (low bush cranberries). In the 1840s Lavrentii Zagoskin (1967, 256) found the people of the middle Kuskokwim taking bilberry (or blueberry) juice as a remedy for practically any illness. The Tsimshian treated diarrhea with blueberries (McGregor 1981). As for lingonberries, or V. *vitis-idaea*, the people of the Kuskokwim took them as a treatment for diarrhea, and applied the juice to eyes afflicted with snowblindness (Lantis 1959, 6, 15). The Inupiat sometimes mashed the berries to a paste and applied them as a poultice around the neck of someone with a sore throat. The crushed berries, mixed with seal or fish oil and then boiled, were administered to stimulate the appetite in a person who had been losing weight (Mauneluk Cultural Heritage Program 1976, 40, 51–52). Around Cook Inlet the Indians heated the berries, wrapped them in a cloth, and applied them as a poultice for headache, sore throat, or a swollen area of the skin (Kari 1981, 41). Other Athapaskans used them to relieve an upset stomach, or morning sickness. The boiled berries were sometimes rubbed on a measles rash (Hall 1979).

For skin diseases Blaschke (*in* Krause 1956, 284) reported that the Tlingits washed with an effusion prepared by boiling a kind of laurel he calls *Kalmia glauca* (probably *K. polifolia*, or bog laurel) (Hultén 1968, 721).

Gentianiaceae (Gentian family)

Plants from the gentian family, scattered all over the world, have been known since antiquity for their healing virtues; in fact, “gentian” is said to be named for a king of Illyria (2nd century B.C.) who used it for the suppression of fevers. In later times the plants were employed primarily as a tonic or bitter, or for the treatment of hysteria, fevers, jaundice, scrofula, or the itch (Le Strange 1977, 125). In America the buckbean, *Menyanthes trifoliata*, was known to be a powerful emetic and cathartic, but when dried it served as a stomachic and tonic (Millspaugh 1974, 514). Others thought it helpful for intestinal worms, fevers, and amenorrhea. It was official in the *NF* until 1926. American Indian groups prescribed this plant for hemoptysis and internal diseases (Vogel 1970, 286).

The Aleuts made a tonic from the roots of the buckbean, with which they treated gas pains, constipation, and rheumatism (Bank 1953). The Tlingit had some medicinal uses for the buckbean, but details are lacking (W. H. Evans *in* Smith 1973).

Scrophulariaceae (Figwort family)

The figwort family is comprised principally of herbs and small shrubs, some of which are saprophytic. Several species had medicinal uses, but the most renowned member of the family in medical circles is the purple foxglove, or *Digitalis purpurea*, source of the essential cardioglycoside. The name *Scrophulariaceae* itself is derived from the fact that the deep-throated nature of the flowers once suggested that they would be helpful in throat infections, including scrofula.

The only representative of this family used medicinally in Alaska was *Mimulus guttatus*, or yellow monkey flower, a root infusion of which was employed by the Chugach for constipation (Birket-Smith 1953, 117).

Plantaginaceae (Plantain family)

The plantain family is a small one, but with a worldwide reputation for its healing powers. *Plantago* species were used in ancient India as a demulcent, among the Romans for application to wounds, and in

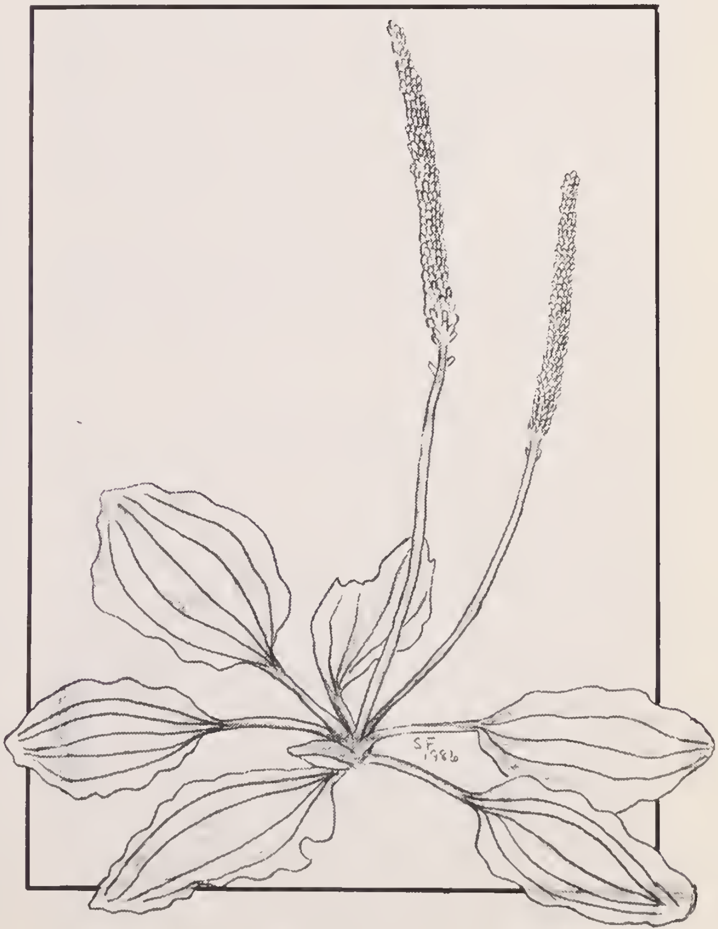

Figure 23. ***Plantago major:*** **common plantain**

later centuries by Renaissance surgeons to promote healing in wounds or as a specific for fevers (Wheelwright 1974, 37, 104, 116). *Plantago* is the source of psyllium seed, which is widely used today as a type of bulk laxative (Billups and Billups 1987, 485). Among American Indians the wild plantain was taken internally for gastrointestinal upsets and as a snakebite remedy. Sometimes the leaves were applied externally to reduce fever, or as a dressing for burns (Vogel 1970, 101, 175; Moerman 1977, 142).

Of the seven species of *Plantago* in Alaska, two have found use for healing. In the Aleutians *P. macrocarpus* root was made into an infusion and drunk as a general tonic (Bank 1953). The Koniag wrapped the leaves of *P. major*, or common plantain, around sore, cracked feet as a soothing dressing (Graham 1985, 66).

Rubiaceae (Bed-straw family)

The Rubiaceae, also known as the madder family, consists of herbs and small shrubs, largely found in tropical or subtropical regions. Two members, both of South American Indian origin, have great medical importance even today: the cinchona tree, the bark of which was the original source of quinine; and *Cephaelis ipecacuana*, from the roots of which ipecac is prepared. The genus *Galium* was used in medieval England as a tonic or as an application to wounds (Le Strange 1977, 47). Recently it has been found to contain the vitamin K antagonist coumarin (Lewis and Elvin-Lewis 1977, 192). Among the North American Indians a member of the genus *Galium*, called cleaver's vine, was taken internally for gonorrhea, fever, and headache. Externally it was popular for skin diseases such as eczema and ringworm (Vogel 1970, 213, 219; Moerman 1977, 79).

In Alaska one species from this family has reputed medical value. The Tanaina made hot packs from *Galium boreale* and applied them to body aches and pains (Kari 1981, 128).

Caprifoliaceae (Honeysuckle family)

The honeysuckle family includes various shrubs and vines of the Northern Hemisphere, among them the honeysuckles, woodbines, elders, eglantine, and highbush cranberries. *Sambucus* (elder) extracts were esteemed in Roman times and in medieval Europe for many medicinal purposes, and the fruits and flowers were listed in the *NF* as late as 1947 for use as a diaphoretic, diuretic, and cathartic (*Merck index* 1983; Le Strange 1977, 222). Most species of elder, however, contain toxic alkaloids and even a form of cyanide (Lewis and Elvin-Lewis 1977, 4, 18). Preparations from the hawthorn (genus *Virburnum* or *Crataegus*) also have found multiple medicinal uses, for example for sores, urinary stones, uterine hemorrhage, and dropsy (Vogel 1970, 314–315). A *Viburnum* derivative was official in the *USP* until 1926 and in the *NF* until 1960, for use as a uterine antispasmodic. This genus also has significant toxic potential, containing coumarin glycosides and viopudiol, the latter a strong antispasmodic which in larger amounts can cause hypotension and bradycardia (Lewis and Elvin-Lewis 1977, 20; Viereck 1987, 35).

The American Indians had multiple medicinal uses for these plants. They employed elder for toothache, inflammations, headache, chest congestion, and swollen breasts, to name but a few of the applications. Others used the hawthorn for dysentery, cramps, general debility, as a diuretic, and as a remedy for "female disorders" (Vogel 1970, 301–302, 314–315).

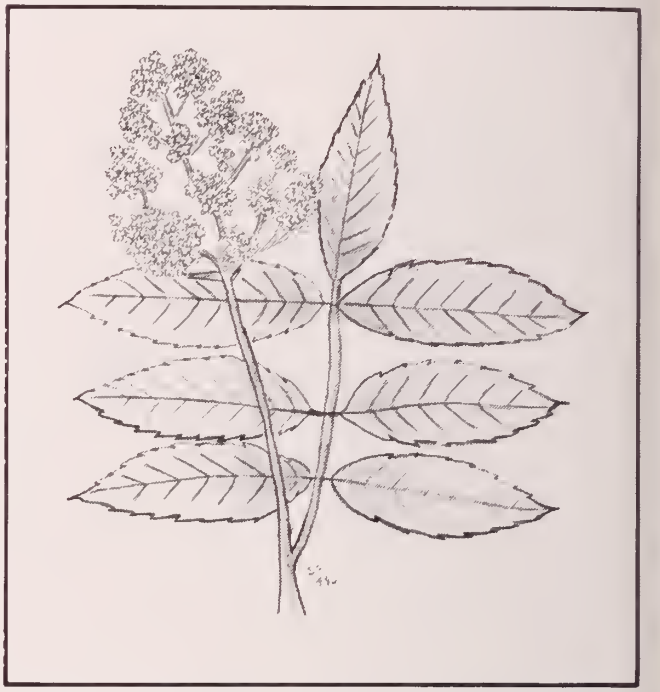

Figure 24. ***Sambucus racemosa:*** **Pacific red elder, elderberry, red-berried elder**

In Alaska the red-berried elder, *Sambucus racemosa*, was employed by the Chugach as a switch in the steambath for treating rheumatism (Birket-Smith 1953, 117). The Tanaina used a tea made from the stem bark externally for skin infections. For the treatment of fever, colds, influenza, or tuberculosis, they peeled off the outer bark of the roots and prepared a tea from it (Kari 1981, 66–67). The Koniag were said to dry the flowers and berries, boil them, and drink the tea for colds, chills, or cramps (Graham 1985, 104; Preston 1961).

The high bush cranberry, *Viburnum edule*, was used chiefly around the north Pacific rim. An infusion of the leaves was considered a good sore throat remedy among the Chugach (Birket-Smith 1953, 117). They prepared another type of infusion, with laxative properties, from cambium shavings, and used a poultice made from the cambium layer for application for two or three days to boils or infected wounds. The fruit, either raw or made into a syrup, was considered helpful for cough or sore throat (Wennekens 1985, 51–52). Around Cook Inlet an

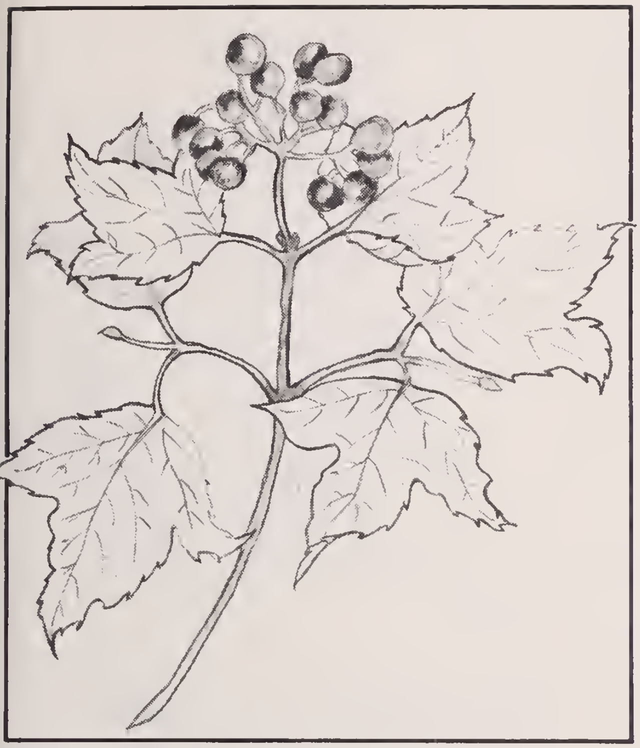

Figure 25. *Viburnum edule:* high bush cranberry

infusion from the bark was used for stomach trouble, sore throat, colds, and laryngitis (Kari 1981, 39).

Valerianaceae (Valerian family)

The valerian family includes herbs and shrubs found mainly in the north temperate regions of the world. The so-called spikenard of the Bible is a form of valerian and was used both as a scent and as a medicine by ancient cultures of the Mediterranean (Wheelwright 1974, 66). In medieval times Chaucer mentions it as a plague remedy (Le Strange 1977, 12). The plant has been popular in Europe and America ever since, mainly for its sedative effects on the nervous system. The active principle is the monoterpene valepotriotes (Lewis and Elvin-Lewis 1977, 168). At the present time two prescription drugs containing valerian are available in the United States (Billups and Billups 1987, 646). The North American Indians took valerian extracts for hemoptysis, pain, and intestinal worms, and made it into a poultice for wounds (Moerman 1977, 197–198).

In the early nineteenth century Dr. Georg von Langsdorff (1814, 2:134) noted that among the Tlingit "all severe diseases are ascribed to the sorcery of their enemies, and the root of a particular species of valerian is considered as the most effectual remedy that can be administered." What Swanton ([1908], 446) called "smelling medicine" is also thought to be valerian and was rubbed on the body for any kind of illness. Around Yakutat the plant was mashed to a powder and applied to a woman's breasts to assist in weaning, since the powder had an exceedingly unpleasant taste. When ground up and soaked in water it could be applied to sore muscles (De Laguna 1972, 658). The Eskimos of St. Lawrence Island, over a thousand miles away, used the plant (*Valeriana capitata*) as a remedy for stomach trouble (Young and Hall 1969).

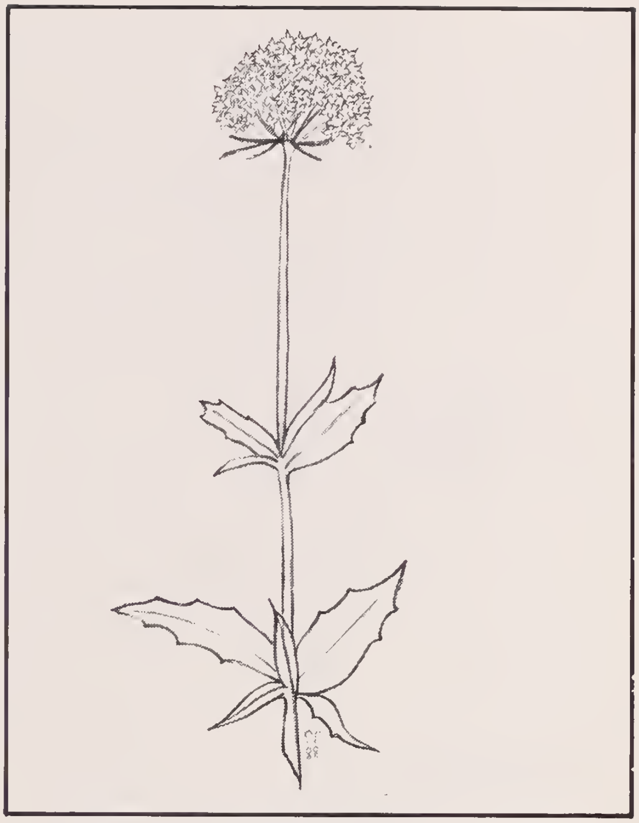

Figure 26. *Valeriana capitata:* valerian

Compositae (Composite family)

The composites are by far the largest family of plants around the world, with well over 20,000 species. Most are herbaceous, but there are also shrubs and a few small trees. Among the well-known members of the family are the sunflowers, thistles, chicory, ragweed, lettuce, artichokes, and the ever-present dandelion. Many medicinal uses have evolved for this group and it would be impossible to do more than hint at a few here, particularly of the plants similar to those found in Alaska.

The oil of *Erigeron*, or fleabane, once found a place in the *USP* as an astringent and diuretic. Yarrow (genus *Achillea*) was listed in the *USP* as a drug for controlling uterine hemorrhage, and has also been employed as a diaphoretic and stimulant (Reynolds and Prasad 1982, 670). Its botanical name refers to the Homeric hero Achilles, who supposedly made an ointment from it to heal wounds (Le Strange 1977, 4). Members of the genus *Matricaria*, or chamomile, are the source of well-known popular remedies, especially for intestinal complaints, and a substance derived from chamomile, alpha-bisabolol, has been shown to have some anti-inflammatory and antifungal activity (Reynolds and Prasad 1982, 678). The tansy (genus *Tanacetum*) has had a long history of use as a carminative tonic, sedative, and as a treatment for gout. An *Artemisia* species was known to Hippocrates, who recommended it to promote uterine evacuation and for disorders of the brain. Another *Artemisia* was wormwood (absinthe), with a long history of use for intestinal worms, fevers, gout, scurvy, and urinary stone (Millspaugh 1974, 319–344; Le Strange 1977, 34). *Artemisia* extracts have definite toxic potential. The plant is the natural source of camphor, for example, and also contains santonin, which can cause headache, vertigo, nausea and vomiting, diarrhea, and seizures. An excessive intake of absinthe may cause a similar picture, called absinthism, characterized by restlessness, tremors, vertigo, vomiting, and ultimately convulsions (Reynolds and Prasad 1982, 105, 317). Species of *Senecio* were popular in ancient and medieval times for toothache, jaundice, stomach trouble, and scrofula, to name only a few of the indications (Le Strange 1977, 230–231). Some species contain a substance called platyphylline acid tartrate, which has an action similar to atropine, although less potent. An extract known as senecionine has some antineoplastic effect but is also a potent hepatotoxin (Reynolds and Prasad 1982, 309, 1753; *Merck index* 1983, 1215).

Among American Indians fleabane was widely used for diarrhea, colds, headache, vaginal discharge, and menstrual troubles. Yarrow was taken internally for stomach trouble and fevers, and applied externally for wounds and rashes. Likewise, *Artemisia* could be taken as a medicinal or used for a wrapping around a bleeding wound. Others employed it for menorrhagia or as a pain remedy (Vogel 1970, 238, 305, 396–397). *Senecio* species were taken internally to stop uterine hemorrhage or to cause abortion (Millspaugh 1970, 358).

In Alaska the Compositae include some of the most popular Native remedies.

Two species of the genus *Aster, A. sibiricus* and *A. subspicatus*, were thought to have medicinal value in the Prince William Sound area. The former, the Siberian aster, was used in times past, but the exact mode is now obscure. The root of the other species, known as the purple daisy, was cleaned and boiled to make a tea that was used to combat colds and fever. The Chugach also prepared an infusion from the roots of *Erigeron peregrinus*, the coastal fleabane, for the treatment of colds and pneumonia (Wennekens 1985, 52-53).

Members of the genus *Achillea*, or yarrow, were used medicinally throughout Alaska. The range of its purported virtues extends to nearly every type of sickness or injury. The Aleuts applied the crushed leaves of *A. borealis* to a bleeding cut or stuffed a rolled-up leaf into the nose to stop a nosebleed. For internal use they prepared an infusion from the leaves and administered it for colds, sore throats, stomach pains, and tuberculosis (Bank 1953). On Kodiak Island an infusion of the leaves was found helpful for asthma, hemoptysis, and hangover (Graham 1985, 29). Around Prince William Sound pulverized yarrow root was applied to boils (Birket-Smith 1953, 117). The Chugach also drank a tea prepared from the leaves for chest congestion and colds, and gargled with it for sore throat. Further, they dried the leaves, stored them in a cloth bag, and then warmed the bag and applied it to aching joints. When the dried leaves were eaten, they were said to aid in uterine evacuation and to enhance the flow of milk after parturition. The leaves themselves, when moistened, served as hot packs for sores, pimples, and boils (Wennekens 1985, 53). The sole use recorded for the more northern Eskimos is by the Inupiat on the North Slope, who prepared an infusion from the dried plant, but the exact way in which it was employed is obscure (Anderson 1939).

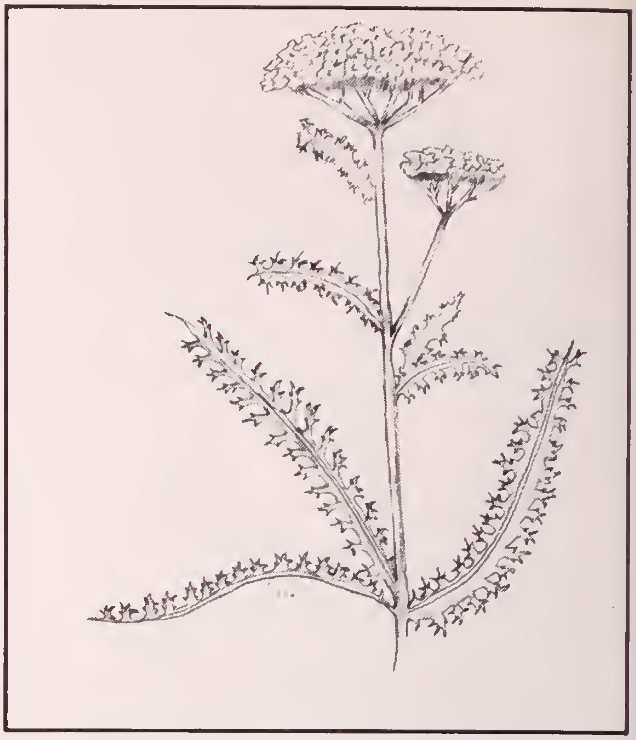

Figure 27. ***Achillea borealis:*** **yarrow**

Among Indian groups yarrow also was highly esteemed. The Tanaina applied cooked or raw, wet leaves as hot packs for earache, sores, or other painful areas. They were also thought to be useful for kidney trouble or stuffed sinuses. The dried and pounded leaves could be applied to sores, cuts, and burns. An eyewash was made by boiling or soaking in hot water the part of the plant which extends above the ground (Kari 1981, 122). Around Yakutat the Tlingit steamed yarrow bark over rocks and placed it over the abdomen as a treatment for amenorrhea. Leaves were heated in the same way for rheumatism or menstrual cramps. When the plant was boiled and placed on a swollen infected area, it was said to draw out the pus. A similar preparation could serve to bathe sore eyes (De Laguna 1972, 656).

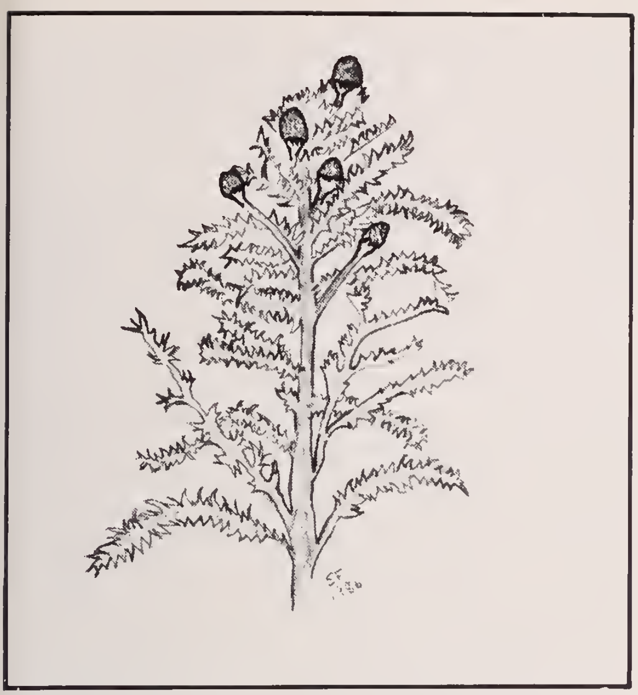

Figure 28. ***Matricaria matricarioides:*** **pineapple weed, wild chamomile**

Matricaria matricarioides, or the pineapple weed (sometimes called wild chamomile), was, according to Hultén (1968, 889), introduced from Europe. Be that as it may, the plant has found widespread use as an herbal remedy among the Alaska Natives. In the Aleutians an infusion made from the leaves was considered a good general tonic (Bank 1953). On Kodiak Island the Natives boiled the whole plant, strained off the liquid and administered it to both mother and baby after childbirth, to serve as a gentle tonic and to promote flow of the mother's milk (Graham 1985, 27). Around Cook Inlet the decoction had a similar use and was also considered a good laxative and useful eyewash (Kari 1981, 124–125). The Chugach also found it helpful as a laxative, if eaten raw (Wennekens 1985, 54). The Yupik ate the tops of the plants raw for hemoptysis, and the dried and cooked seed-heads for colds and indigestion (Ager 1980; Oswalt 1957).

Probably no single plant has had a wider range of medicinal uses in Alaska than *Artemisia tilesii*, or closely related species. Just as the devil's club was the panacea in southeastern Alaska and along the north Pacific rim, the stinkweed (also called sage, or wormwood) was the panacea of the Eskimo peoples.

Stinkweed was one of the few plant remedies of the Inupiat. They treated injuries and swellings with a poultice made from the dried plant, and dried and boiled the plant as a cold remedy (Anderson 1939). In the Noatak River region an infusion from the leaves was considered a useful treatment for colds or hemoptysis (Lucier *et al.* 1971). Around the Bering Strait the plant was particularly esteemed for sores that failed to heal. In the eastern Norton Sound area stinkweed was used for many types of illness, but especially for gas pains (Lantis 1959, 5, 18). Around Kotzebue Sound the Eskimos wrapped the leaves directly around sores, boils, aching joints, or other painful areas, or after heating them applied them to an aching ear. Either fresh or dried, the leaves were boiled into a tea, which was given for colds, bad lungs, bladder trouble, or aching bones (Mauneluk Cultural Heritage Program 1976, 12, 27–28, 40, 53). On the North Slope the people prepared an infusion for the treatment of colds or pneumonia. An obviously more recent application is a combination of stinkweed and soda to relieve indigestion. The liquid obtained by boiling the plant was sometimes applied as a compress for skin rashes or open sores (DeLapp and Ward 1981, 24, 43, 46, 54, 56).

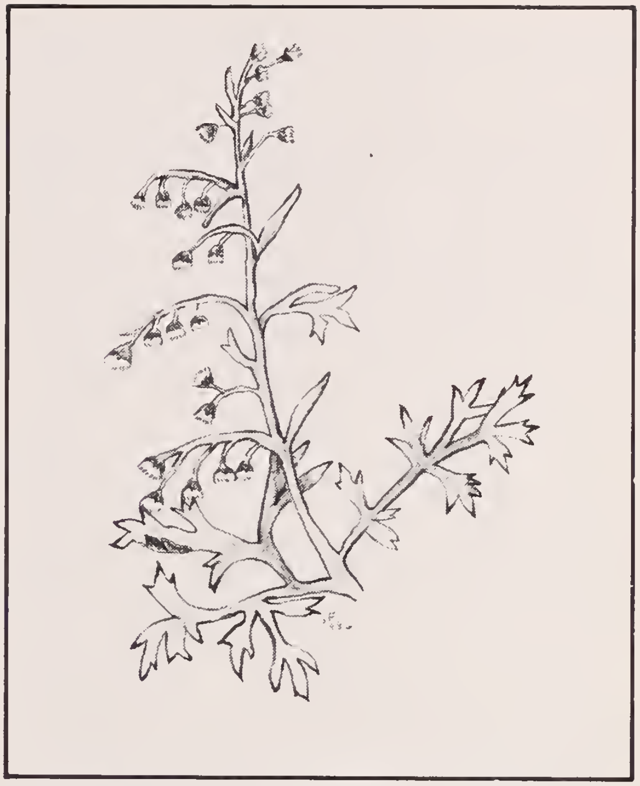

Figure 29. ***Artemisia tilesii:*** **stinkweed, sage, wormwood**

Among the Yupik *A. tilesii* was also a very popular remedy. An infusion of the leaves was taken for arthritis, while an infusion of the seed-head was considered good for constipation and as a general tonic. Breathing the vapors of the boiled plant was thought to relieve chest congestion, and boiling the stems produced a medicine for swollen areas. The leaves were applied externally as a poultice to large cuts to control bleeding or to treat infection (Ager and Ager 1980). When heated they helped relieve joint pains. The Yupik also used the plant as a switch in the steambath for the treatment of sprains or sore limbs (Lantis 1959, 5). On the Kuskokwim the people rubbed the juices of this plant on the hands as a deodorant; a similar practice using the stems and leaves has also been reported for the people of St. Lawrence Island (Ager and Ager 1980; Young and Hall 1969).

The Koniag also found multiple uses for the plant. They prepared a tea from the leaves, which was taken as a blood tonic or as a remedy for varicose veins. The extract from dried or freshly boiled leaves was applied externally on infected cuts or sores. The people chewed the leaves to combat colds or influenza, or they heated them for use as a poultice for arthritis or other painful areas. The seeds were said to be good for heart trouble (Graham 1985, 17–18). The Chugach drank an infusion made from the leaves for an upset stomach or for rheumatism (Wennekens 1985, 54).

The Aleuts, like the Yupik, used the plant as a switch in the steambath, after heating it on hot rocks. The warmed leaves could also be applied as a poultice for painful areas. An infusion of the leaves was thought to be especially helpful as a tonic for dying persons (Bank 1953).

Among the Athapaskans around Cook Inlet multiple medicinal uses of the plant also emerge. Except for an infusion of the leaves taken for colds, all reported preparations were for administered externally. The leaves were ground up and applied over a layer of grease on a burn. The liquid resulting from boiling the leaves was applied as a poultice for toothache, earache, or snowblindness. Such a preparation was also rubbed on a pregnant woman's abdomen before a healer attempted an external version. The water was also applied to rashes, sore eyes, cuts, athlete's foot, and inflamed joints. Crushed raw leaves were also considered helpful to itching areas of the skin. Whole leaves were sometimes wrapped around a cut as a dressing (Kari 1981, 126–127). As an abdominal bandage after a steam bath, it was thought to break up colds (Osgood 1937, 116). The Tanaina, too, used the plant as a switch in the steambath, particularly for rheumatism (Kari 1981, 127).

Finally, there is an early record of the Tlingit having used a type of *Artemisia*, called *A. vulgaris* by Blaschke (*in* Krause 1956, 283), for the treatment of pleurisy.

The coltsfoot, or the genus *Petasites*, found medicinal use around the north Pacific rim. According to Osgood (1937, 116) the Tanaina Indians prepared a tea from the roots of *P. palmata* [sic] to "make the blood soft," and chewed the root to stop hemoptysis. (Hultén [1968, 914] states that *P. palmatus* has not been described from Alaska.) The Tanaina also soaked the root of *P. hyperboreus* in hot water and drank the infusion for tuberculosis, chest trouble, sore throat, and stomach ulcers (Kari 1981, 130). Their neighbors the Chugach chewed the root for chest congestion and colds (Wennekens 1985, 54).

Senecio congestus, known as marsh fleabane, was thoughtby the Yupik to be poisonous and had no recorded medicinal use (Lantis 1959, 17). The Aleuts, however, applied the leaves of another species, *S. pseudo-arnica*, to cuts and boils to promote healing (Bank 1953).

The leaves of a dandelion, *Taraxacum officinale*, was steamed by the Aleuts and applied to indolent ulcers (L. M. Turner *in* Smith 1973).

As a final note, Dr. Robert White in 1880 reported that the Tlingit pounded the leaves of a *Tanacetum*, or tansy, mixed it with bear gall, and applied it to joints for the treatment of arthritis (White 1880). According to Hultén, this genus does not occur in Alaska.

Unidentified Plants

At times the source materials are too vague to permit a precise identification of the plant in question. This situation may arise because of the observer's inadequate knowledge of botany, or of the language, or because the informant has been unable to locate a specimen for direct examination. Occasionally an early writer has made use of obsolete terminology.

An eighteenth-century Russian navigator noted that the Aleuts applied a yellow root to wounds, accompanying the treatment with fasting (Ocheredin *in* Coxe 1787, 201). According to Veniaminov (1984, 292–293) they prepared an infusion of bitter herbs for fever, and treated swellings and muscle pains by the application of bitter roots baked before the fire.

Among the Koniag a plant called *chyvykhyat'* was used raw for the relief of constipation resulting from the immoderate consumption of raspberries. An infusion of the roots of this plant was also considered an excellent laxative. The roots of another plant, called *chikinalyakhpik*, were boiled and the bitter and unpleasant infusion given twice a day for the relief of internal boils. The roots of *taganak*, when crushed and steamed on a hot rock, were applied to swollen areas (Black 1977; Pierce 1978, 129–130). On the island of Sutkhum the Koniag dug a root called *shishkuk*, the leaves of which they smoked, whereas they administered an infusion of the leaves to pregnant women (Davydov 1977, 179).

The Chugach applied the hot leaves of a certain plant to aching ears. For hemoptysis they drank an infusion of a pulverized plant not otherwise identified, and for diarrhea they took charcoal mixed with water (Birket-Smith 1953, 117).

The Yupik Eskimos of the lower Kuskokwim area used a short plant with yellow flowers to make a pregnant woman bleed, presumably as a means of inducing abortion (Lantis 1959, 34).

In northern Alaska several plants were not fully identified in the study by DeLapp and Ward (1981, 22, 24, 29–30, 48, 52). Some "special green leaves" were placed over and under a cancer victim for several days as a form of treatment. For a cold, or pneumonia, a bag of plants soaked in hot water was placed on the chest, and a similar technique, perhaps with a different plant, was used for aching bones. Crystallized wood sap, or alternatively wood-shavings, were applied to open wounds to prevent infection and promote healing. For generalized pain a certain plant was wetted and applied to the skin, although if allowed to remain too long, it could cause a bad skin rash. Nicholas Gubser (1965, 239–240) mentions that the inland Eskimos boiled "one or two leaves" as treatment for backache and sore joints.

Several unidentified medicinal plants are reported for the Athapaskans. Joan Townsend (1965, 346) described a plant the Tanaina called *nakaxetsa*, which was said to have large leaves and grow beside creeks. Either the whole, raw leaf or the mashed root pulp was applied to cuts, boils, and sores, and was said to have an anesthetic effect. For tuberculosis the Tanaina prepared a tea from the leaves of a three-foot tall bush called *shentona*. The water from the boiled leaves of another plant was used as an eyewash for any type of eye affliction, including snowblindness. Another Tanaina remedy of uncertain identification was known as *nkedu'i*, the root of which was chewed for colds, sore throat, and tuberculosis (Kari 1981, 132).

Among other Athapaskan groups, The Peel River Kutchin made a tea of some type for kidney trouble, and smashed and boiled a certain root as a laxative (Osgood 1936, 93). Members of the Upper Tanana band boiled and drank the ashes from burning wood punk as a treatment for tuberculosis (McKennan 1959, 109).

In southeastern Alaska Dr. Blaschke (*in* Krause 1956, 283) described the use by the Tlingit of *Cornicularia richardsonii*, a plant of uncertain classification, which was boiled and administered as an infusion for inflammation of the lungs. Finally, Swanton lists a few plants which are obscure. "Land-otter medicine" (*ku'cta nak*) was taken by someone who was "short-winded in one place" [sic]. "Seal's tongue" was a large flower that was chopped up and applied to boils or open sores. Certain berries called *cax wa s!i* grew in strings. When the vines were broken and the ends brought close to a white spot in the eye, the lesion was said to have come out. Taking another plant, called *ca'xnasti nak*, or syphilis medicine, had to be preceded by a drink of the patient's own urine (Swanton [1908], 446–447).

WHAT PLANTS WERE USED FOR HEALING?

Hultén (1968) lists 89 families and 421 genera of vascular plants found in Alaska, not to mention hundreds of individual species and subspecies. These totals, of course, do not include the many Alaskan families and genera of algae, fungi, lichens, and true mosses, which are also generally classified as plants.

Among the vascular plants 41 families (46.1% of the total) include members which are recorded as having medicinal use by the Alaska Natives. Of the 421 genera of Alaskan vascular plants, some 78 (18.5%) are known to have been used medicinally. Considered from another perspective, in the 41 plant families with medicinal use in Alaska, there are 268 genera, of which 78 (29.1%) were used by the Alaska Natives for healing.

These figures are only crude estimates, of course, of

Table 1. ***Reported use of medicinal plants, by Alaska Native cultural group***

Cultural group	Identified genera vascular	Identified genera non-vascular	Unidentified	Total genera
Tanaina	40	2	3	45
Tlingit	30	4	4	38
Chugach	29	4	2	35
Yupik	19	2	1	22
Aleut	18	2	3	23
Koniag	14	1	4	19
Inupiat	12	1	5	18
Tsimshian	8	1	—	9
Ingalik	6	—	—	6
Kutchin	5	—	1	6
Upper Tanana	3	2	1	6
Haida	2	—	—	2
Eyak	2	—	—	2
Han	1	—	—	1
Koyukon	1	—	—	1

the extent of plant use. An analysis by individual species is not feasible, because often the source materials do not provide a species or subspecies identification. Furthermore, approximately two dozen plants remain unidentified, even with respect to family, although some of these likely fall into known categories of healing plants.

Some idea of individual plant use may be gained, however, by examining the available data by genus. There are 15 cultural groups for which some information on medicinal plants is presented in this paper. Of these, the genus *Picea* was used by twelve, *Rumex* by eight, *Echinopanax* by seven, *Artemisia*, *Ledum*, and *Achillea* by six, *Salix*, *Vaccinium*, and *Matricaria* by five, and *Ribes*, *Veratrum*, *Juniperus*, and *Heracleum* by four cultural groups each. If the unidentified plants are excluded, some 45 genera were prescribed by a single group only.

Medicinal plant usage by cultural group is somewhat misleading, since the information available is directly dependent on the intensity and depth with which each group has been studied. The figures shown in Table 1 probably have some value, however, in demonstrating that the use of medicinal plants is correlated with their availability. Those areas of Alaska that have a greater rainfall and a milder climate are known to have a richer variety of plant life. It is not surprising, therefore, that the aboriginal peoples of these areas have made greater use of plants in their healing tradition.

WHAT WAS TREATED BY PLANT REMEDIES?

An analysis of the diseases and conditions which the Alaska Natives treated with plant remedies is fraught with difficulties. As already pointed out, the source materials are mostly written by persons untrained in medicine, and their use of terminology is often neither consistent nor precise. Of even greater importance, perhaps, is the terminology of the informants themselves, few of whom had any knowledge of the Western medical tradition, and often thought of diseases in different terms. When information was translated from one language to another, even greater confusion could result. Finally, differing perceptions by informants, chroniclers, and physicians as to what exactly constitutes a disease, as opposed to a symptom or sign, further obscure the issue.

Despite these problems, a few observations might be in order. Certain symptom complexes and illness types seem to be especially prominent. Most represent the common sicknesses and injuries of everyday life and suggest that these conditions were widespread, presumably even before European contact in the eighteenth and nineteenth centuries (Fortuine 1985/86). Moreover, it is significant that these symptoms and signs, although annoying, are rarely life-threatening or disabling. In generations past the more serious conditions were referred to the shaman for magical forms of treatment, whereas today they are usually promptly referred, even by the Native healers, to hospitals and clinics.

Within the infectious disease group, respiratory ailments seem to have been particularly common. Disease boundaries among these conditions are often blurred, and they are here divided for convenience into three principal categories. Plant remedies for upper and lower respiratory infections (colds, influenza, cough, chest congestion, pleurisy, and pneumonia) are known for every major Alaska Native culture. Some 29 plants, here defined at the genus level, are described in this paper, the most popular ones being spruce (five cultures), and Labrador tea, stinkweed, and devil's club (four cultures each). For sore throat and sore mouth, the Natives used at least 17 medicinal plants, particularly the juniper, which was reported for three groups. Herbal therapies for tuberculosis (including consumption and hemoptysis) totalled 31, with spruce and devil's club each used by four cultures. This large number of treatments, according to an old medical adage, suggests that none of the remedies was very effective.

Nine plants are recorded for the treatment of venereal disease, usually syphilis, with only one plant (devil's club) used by two cultures. Most of the known remedies are from either the Koniag or the Tlingit, both of whom suffered greatly from the disease in the eighteenth and nineteenth centuries (Fortuine 1985a). Diarrhea remedies were found in every major Native culture and included 12 genera, only two of which, sourdock and *Vaccinium* berries, were used in more than one part of Alaska. Some 30 plant remedies are recorded for boils, skin sores, or infected cuts, all of which were known to be common at the time of contact. For these conditions the most widely used plants were spruce (four cultures) and yarrow (three cultures).

A total of 25 types of plant were prescribed for cuts, wounds, and external bleeding, the most popular ones being spruce, used by four cultures, and three other plants used by two cultures each. For burns, another common injury, seven plants are described, with only spruce having two advocates.

A wide selection of plant remedies were prescribed for chronic disease, pain, and physiological upsets. Twenty different genera were valued for their laxative properties. Four cultural groups employed devil's club for this purpose, while two each used fireweed, pineapple weed, or iris. Six plants were known particularly for their emetic properties, one of these, devil's club, by three separate cultures, and sourdock by two. For the symptom complex of stomach-ache, ulcer, gas, and indigestion, some 25 medicinal plants are described, the most popular ones being Labrador tea, endorsed by three groups, with alder and sourdock each recommended by two.

For non-specific or internal pain (including stomach pain and headache), the list of remedies swells to 26, with stinkweed advocated by four cultures, and seven other plants by two each. Fifteen plant genera were employed for arthritis, rheumatism, and non-specific joint pains. By far the most popular was stinkweed, recorded for no fewer than five major cultures. Devil's club had three advocates, whereas cow parsnip and yarrow had two advocates each. Thirteen plants found application for the treatment of skin rashes, with a single overlap, stinkweed, used by two groups. Finally, 21 genera were employed by various Native groups for eye problems ranging from snowblindness to cataract. The most popular plant was the currant (*Ribes spp.*), used by three cultures, with four other plants favored by two each.

The most striking conclusion to be drawn from the foregoing is that, although the Alaska Natives found medicinal virtues in many plants, only a few, notably stinkweed, devil's club, spruce, yarrow, and Labrador tea, were more than purely local remedies. Some other plants, such as willow, sourdock, and wild celery, were used medicinally over a wide area, but generally for different indications in different cultures. The rest were used by only one or perhaps two cultural groups.

Whether significant cultural diffusion occurred is difficult to judge. We have already seen that plants of the same family have been found useful as medicines in widely separated parts of the world, with no possibility of other than independent discovery. In Alaska there are certainly similarities of usage in broad geographical areas (for example the Aleutians and Kodiak Island, Cook Inlet and Prince William Sound, or the Yukon-Kuskokwim Delta and Bering Straits region), but these patterns may be the result of similar geography and climate, and therefore plant distribution, rather than sharing between cultures. In most parts of Alaska, in fact, adjacent cultures were at odds with each other to the point of occasional open hostility.

ALASKA MEDICINAL PLANTS IN TODAY'S WORLD

Alaska traditional plant remedies continue to be held in high regard by the Alaska Native people. Although the tradition has been continuous, with adaptations to new diseases and newly available plant substances, herbal remedies have in recent years received more public attention than formerly. This new focus can be partly explained by the recent resurgence of popular interest in "natural" foods and "holistic" medicine throughout the nation. An even more important factor, however, has probably been the new cultural pride and self-awareness of the Alaska Native people themselves following the passage of the Alaska Native Claims Settlement Act in 1971. Among the available studies of Alaska healing plants, at least four trace their origin directly to this renewed interest in preserving cultural traditions (Mauneluk Cultural Heritage Program 1976; Hall 1979; DeLapp and Ward 1981; Kari 1981). In one notable case (DeLapp and Ward) the Native group was concerned about the safety of their traditional healing practices.

Do Alaska Native traditional plant remedies have a place in the modern world? The answer is of course "yes." They are in fact being used in all parts of bush Alaska and in some urban settings as well. Many Natives consult their own healers or try their own local prescriptions before visiting a hospital, clinic, or even their Community Health Aide. Now that traditional healing has to a greater extent "come out of the closet" and is admired in the larger society as well, this usage will not only continue but probably also increase.

The next question must be: Do these remedies work? For most, the answer is not clear, nor can the matter be easily settled. Testimonials and anecdotal accounts of cure are not of course in short supply, but these must be viewed very skeptically. All health workers (not excluding traditional healers) know the fickleness of disease, which often takes a totally unexpected turn for better or for worse regardless of medical intervention. The simple fact of administering a medicine, even an inert one, changes the patient's perception of his or her illness, often for the better.

The only means by which these remedies can be properly tested is a controlled, preferably double-blind, clinical trial, but one of this type would be technically very difficult, if not impossible, under Alaskan field conditions. Adequate numbers for statistical validity would be hard to find, and the costs of travel and follow-up nearly prohibitive. Even if other conditions could be met, however, the problem of drug standardization would still be present. Since each plant specimen probably contains a different amount of the active drug principle or principles, depending on subspecies, soil conditions, and climate, even equal amounts of the plant substance would not be equivalent in their effects on the disease process. Furthermore, since the "medication" is not pure, due to other chemical substances present in the plant and different methods of preparing it by different healers, the true therapeutic effects of the remedy in question would be nearly impossible to measure scientifically.

If the active principle could be chemically isolated from the plant, the process of scientific evaluation could be improved somewhat, but there would still be no way to eliminate the necessity of controlled trials. As mentioned in the body of this paper, chemical analysis has been carried out on a few Alaskan plants and certainly on a number of closely related plants found elsewhere in the world. Often a physiologically active substance has been isolated, and in not a few cases this substance has been developed into a standard medication. At this point, however, the philosophical question arises whether such a chemical substance is still a traditional remedy once it has lost its cultural trappings, or whether it has become a part of the Western medical tradition.

Scientifically proven or not, of course, traditional plant remedies will continue to be used in Alaska, simply because many people have confidence in their efficacy. The third question, therefore, must be: Do Alaskan herbal remedies have the potential for harm? The answer is a definite "yes."

Even if the remedy has no physiological effect, and thus does not influence directly the course of the disease, its use may unneccessarily delay definitive diagnosis and effective treatment. For most minor self-limited illnesses the stakes are not high, but for some diseases, such as cancer, meningitis, or myocardial infarction, the outcome could be disastrous.

Even more important than the delay in diagnosis and treatment is the fact that some herbal medicines contain strong metabolic poisons that could cause independent harm or worsen the patient's illness. A partial list of these potentially dangerous substances may be found in Table 2. Other plants, such as valerian and devil's club, have marked physiological effects and in excessive amounts could lead to toxic reactions. Furthermore, crude plant extracts contain many unknown and unstudied chemical ingredients, some of which might have a favorable but others an unfavorable effect.

Table 2. ***Some toxic substances found in Alaska medicinal plants***

Genus	Toxic substance(s)
Anemone	Protoanemonin, elatine
Angelica	Coumarins, (closely related plants contain coniine)
Artemisia	Santonin
Caltha	Aconite
Equisetum	Thiaminase
Kalmia	Andromedotoxin, grayanotoxins
Ledum	Ledol
Oxytropis	Teratogen, locoine (?)
Polygonum	Oxalic acid, tannic acid
Rumex	Oxalic acid
Sambucus	Toxic alkaloids, form of cyanide
Senecio	Platyphylline acid tartrate, senecionine
Urtica	Serotonin
Veratrum	Ester alkaloids, jervine
Viburnum	Viopudiol, coumarin glycosides

It is a popular view today that what is "natural," "organic," or "holistic" is somehow more effective and "better for you" than a product of a tightly regulated pharmaceutical laboratory. "Nature's way" has great emotional appeal for many people in our plastic, packaged contemporary world. The attractiveness of traditional remedies is enhanced by the notion that these medicines are sanctioned by centuries of use and experience by wise persons who have lived close to the land. What such enthusiasts fail to recognize is that most of the drugs used in modern medicine arose in the same way, but today have in addition the inestimable virtue of being chemically pure and scientifically tested for effectiveness and toxicity. As a final point, it should be remembered that as a result of modern science, many healing plants in the Western medical tradition have been officially discarded from the pharmacopoiea, because they have been proven to be ineffective, harmful, or both. Although a similar but informal process has no doubt also occurred in traditional societies, it is unlikely that the information thus gathered has been widely disseminated among other healers, especially of other cultures.

CONCLUSIONS

The plant remedies of the Alaska Natives have had a rich tradition, and many of them will doubtless survive for generations to come. They have flourished because of the confidence that people have in their effectiveness. Moreover, they are readily accessible, inexpensive, and consonant with the cultural tradition of those who use them. Some of the plants have components of known therapeutic value that can be useful in treating specific diseases. Other plants very likely have secrets that have not yet been revealed to science. In any event, most of the remedies have an odor, taste, or physiological effect that the patient (and the healer) associates with cure.

Yet the plant remedies of the Alaska Natives should not be overrated. Although the different Alaskan cultural groups probably discovered the use of these plants independently, only a few of the plants—or closely related ones—have not also been used by American Indians or in the Western medical tradition. Like all drugs, they have limitations and inherent dangers, especially if taken for long periods without supervision. They may hasten recovery in some cases, although in this connection the placebo effect can never be discounted. In other cases they may unnecessarily delay essential treatment by far more effective modern drugs or other modalities. Further, crude plant extracts are impure substances that may contain a varying amounts of several active principles, which themselves may be helpful *or* harmful.

Alaskan herbal remedies will continue to be widely used, whatever their inherent virtues or hazards. I offer the following recommendations to ensure that they will be used in the safest and most effective way:

1. More medicinal plants need to undergo chemical analysis, so that their active principles may be isolated. The purified substances must then be subjected to rigid physiological, pharmacological, and toxicological testing, first in animal models, and later, if warranted, in humans. Such studies may lead to important new infor-

mation not only on the value, but also on the hazards of these remedies.

2. The traditional use of healing plants needs to be further documented and analyzed. Much vital information has already been irretrievably lost and more is in danger of disappearing forever as memories fade and elderly healers die. More studies are needed not only on the identification of plants, but how and why they were used. Such research not only contributes to ethnographic knowledge, but it may also turn up information of value to society at large. It would also be desirable that other Native organizations follow the lead of the North Slope Borough and commission studies on the the possible hazards of traditional remedies in light of modern science.

3. Traditional healers themselves should be encouraged to "step forward" and permit extensive tape-recorded interviews, not only about the plants they use, but also about the way in which they receive their training, diagnose illnesses, and select treatment.

4. Healers should be brought into closer relationship with the health care system. They should be treated as colleagues and permitted and encouraged to receive training in clinical assessment similar to that now received by the Community Health Aides. Training should emphasize the importance of prompt referral in certain kinds of illnesses and injuries.

5. Physicians, public health nurses, and other practitioners need to become more sensitive to the role that traditional healers play in Alaska Native society. They should learn to accept not only that the practitioner exists "out there," but also that he or she has something positive and useful to offer many patients. Uncomfortable as it may be to acknowledge, Natives often have greater confidence in their own healers than in the system of hospitals and clinics. To promote better understanding, Western health practitioners need to be thoroughly and positively oriented with respect to traditional healing. They should meet regularly with Native healers to exchange views, and, if possible, to learn from each other the contributions each can make to the health and welfare of the people of Alaska.

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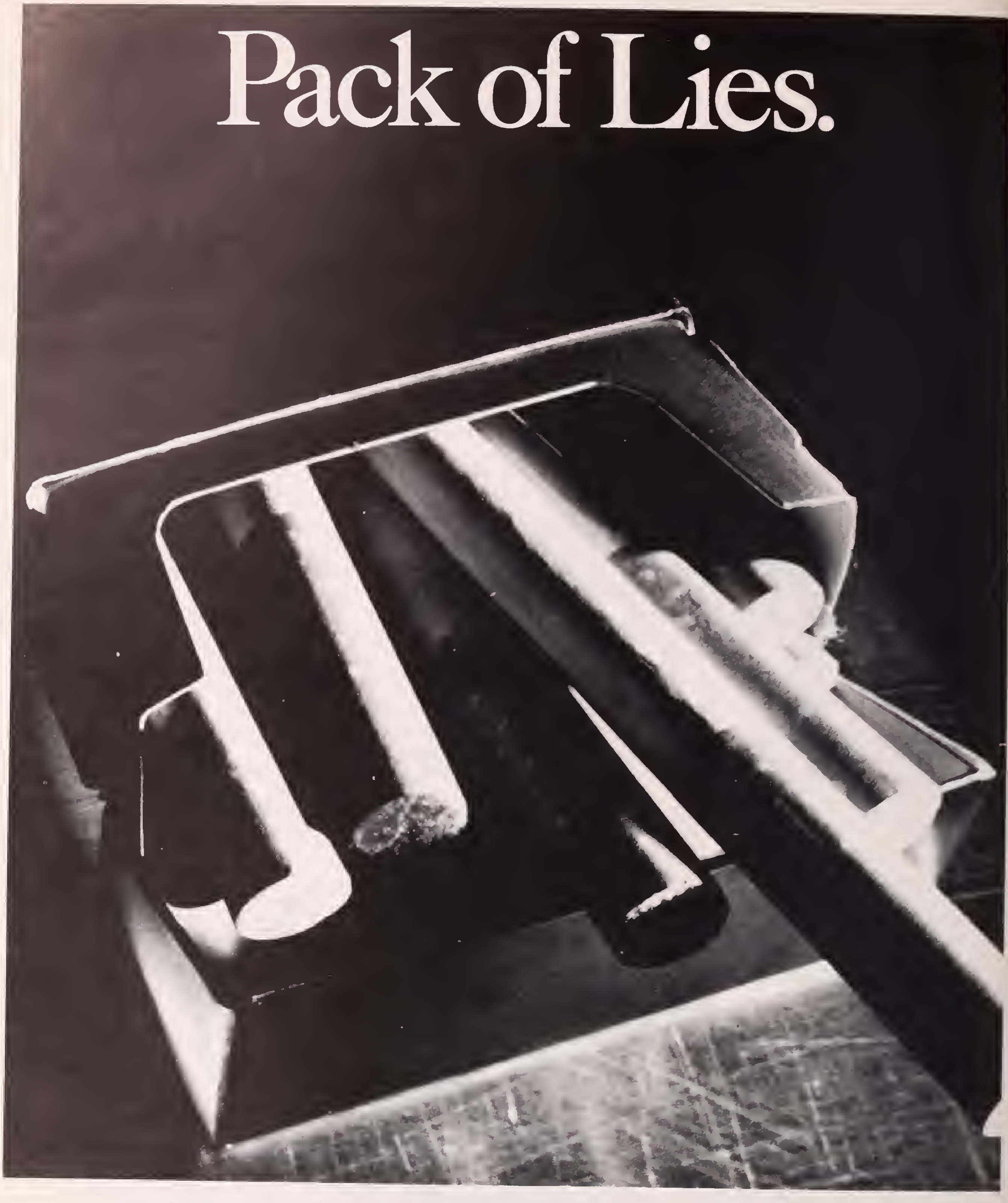

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News from the American Society for Circumpolar Health

The annual business meeting of the American Society for Circumpolar Health (ASCH) was held on October 8, 1988 in Fairbanks, Alaska in conjunction with the Alaska Science Conference sponsored by the Arctic Division, American Association for the Advancement of Science (AAAS). The meeting was well attended; your society is solvent; and your board of directors will be challenged to keep up with the many exciting events planned for the next few years. We all owe particular thanks to Dr. Helen Beirne, our treasurer, for paying meticulous attention to our financial records and to Dr. Frank Pauls, our secretary and chronicler, who somehow keeps us organized.

We shared information at the annual meeting about circumpolar health activities. All were impressed by the widespread enthusiasm that is supporting a broad range of activities.

- Your board is working to establish an endowment fund to support circumpolar health.
- ASCH contributed funds and ideas so that Dr. Jens Peter Hart Hansen, president of the International Union for Circumpolar Health, could attend the XXth meeting of the Scientific Committee on Antarctic Research (SCAR) held in Hobart, Tasmania, Australia, September 5-16, 1988.

Dr. Hart Hansen provided this written report of his activities.

> On the invitation of Desmond J. Lugg, M. D., Chairman of the Working Group on Human Biology and Medicine, I took part in the Working Group Meeting and a colloquium on Human Factors Research in Antarctica, 7-9 September, representing the International Union for Circumpolar Health. Nearly 50 persons attended the colloquium. Ten of the Working Group's 17 members were present. The following countries are represented in the Working Group: Argentina, Australia, Chile, China, France, Germany (BRD), India, Japan, New Zealand, Norway, Poland, South Africa, United Kingdom, USA, and USSR.
>
> During the meeting I had the opportunity to address the Working Group speaking about international cooperation in circumpolar health and arctic medicine, outlining the scope and purposes of the Union and giving factual information about the Union, the adhering bodies, the statutes, and our international congresses. I also had the opportunity to speak about the publications "Arctic Medical Research" and "Alaska Medicine." As conjectural areas of Arctic/Antarctic cooperation were mentioned occupational health hazards, environmental pollution, and indoor air pollution.
>
> There was much interest in the work of the Union, particularly the international congresses and the journals. It was agreed by SCAR that the Working Group will be an affiliated member of IUCH and that the chairman, Desmond J. Lugg, M.D., will take a seat in the Council according to the statutes of IUCH. The individual members of the Working Group and all attending the colloquium will receive information about the forthcoming Whitehorse congress in May 1990 and will be placed on the mailing list of "Arctic Medical Research."
>
> Desmond J. Lugg, M.D., has agreed to be a corresponding editor for "Arctic Medical Research" on Antarctic Medicine and Research representing the Working Group. Personal agreements were made with several of the members of the Working Group with regard to future contributions to "Arctic Medical Research." The Working Group decided to convene in connection with the Whitehorse congress.
>
> The colloquium was highly successful. A number of issues in health care were discussed including health registers, accidents, tourists, alcohol and tobacco usage, infectious diseases, medical screening, and women and families in Antarctica. Among areas of research to be carried out in Antarctica as part of national and collaborative multidisciplinary programs over the next decade were antarctic/space analogous studies, behavioral studies, and cold related studies. An article on the colloquium will be published in "Arctic Medical Research."
>
> During the meeting I took part in the SCAR social arrangements and had the opportunity to meet SCAR executives and other key persons from the many different disciplines which are active in antarctic research. It was my impression that IUCH was recognized internationally and also outside our own field. I am grateful that it was made possible for IUCH to be represented at this important meeting.
>
> Further details about the SCAR meeting and the Working Group meeting are available. Please, do not hesitate to contact me if you have any questions.
>
> A representative for the German Society for Polar Medicine was present in Hobart. The Society was encouraged to apply for affiliated membership of IUCH. Four new members of the American Society for Circumpolar Health signed up.

- The Academy of Sciences of the USSR together with other Soviet scientific organizations is sponsoring a "Conference of Arctic and Nordic Countries on Coordination of Research in the Arctic" in Leningrad, USSR, December 12-15, 1988. The topics to be discussed include results of recent research in the arctic and possible fields of cooperation in the study of this area ofthe earth as well as measures for coordinating such cooperation.

- Dr. Hart Hansen, attending the Leningrad meeting to represent the International Union for Circumpolar Health, will present a paper on "International Cooperation in Circumpolar Health and Arctic Medicine."

ABSTRACT

International Cooperation in
Circumpolar Health and Arctic Medicine

International cooperation in circumpolar health and arctic medicine has improved through the recent decades. Since 1968, international conferences on circumpolar health have been convened every third year organized jointly by the American Society for Circumpolar Health; Canadian Society for Circumpolar Health; Siberian Branch of the U.S.S.R. Academy of Science, Medical Section; and Nordic Council for Arctic Medical Research representing Denmark/Greenland, Finland, Iceland, Norway and Sweden.

These organizations created formally the International Union for Circumpolar Health in August 1981. The objects of the Union are to promote international cooperation in the study of circumpolar health; to encourage and support research and exchange of scientific information in the circumpolar health sciences; to promote public awareness of the current situation of circumpolar health; and to provide a means of communication with other organizations.

The Union covers the whole sphere of circumpolar health and arctic medicine and represents internationally the national bodies of researchers and other interested persons in the field. The union is affiliated with World Health Organization. The publication of the Nordic Council for Arctic Medical Research, *Arctic Medical Research*, is presently the official journal of the Union.

- The SCAR Working Group on Human Biology and Medicine accepted affiliated membership in the IUCH. Dr. Desmond Lugg, M.D., chairman of the SCAR Working Group, will take a seat on the council in accordance with statutes of the IUCH.

- The Division of Polar Programs, National Science Foundation, has begun work on the biennial revision of the U.S. Arctic Research Plan. Current plans call for consultation and workshops from December 1988 to January 1989 with preparation of a revised draft for presentation to the Interagency Research Policy Committee by March 1989. **Now is the time to submit revisions.**

- A circumpolar meeting on **"Tobacco and Health"** will be held, April 4-6, 1989 in Yellowknife, Northwest Territories, Canada. This meeting will be jointly organized by the IUCH and the World Health Organization (WHO). The purpose of the meeting is to bring together health professionals, consumers, local residents, and politicians in order to set a strategy for an efficient, concerted action against tobacco in the Arctic.

- We are already anticipating the 8th International Congress on Circumpolar Health to be held in Whitehorse, Yukon, Canada on May 20-25, 1990.

- Two health scientists were appointed by Governor Steve Cowper to the nine-member board of the newly created Alaska Science Foundation: Dr. Anne Lanier, director, Arctic Investigation Laboratory-Centers for Disease Control, Anchorage (long an active member ofASCH and currently a member of the board), and Dr. Francis Williamson, director of the Institute of Arctic Biology, University of Alaska, Fairbanks, and former commissioner of the Alaska Department of Health and Social Services. We wish you both great success. The ASCH is prepared to assist you at any time.

- ASCH notes the increasing activities of the Alaska Department of Health and Social Services (DHSS) to further international cooperation in the Arctic. Under leadership of Myra M. Munsen, commissioner, DHSS has entered into an agreement with the Ministry of Health, Health and Welfare, Canada to exchange information, expertise, and experts to increase cooperative efforts and to improve the health of Arctic inhabitants. DHSS also has entered into agreements with the Far Eastern Branch, USSR Academy of Sciences; the USSR Ministry of Health; and the Institute for Circumpolar Health Studies, University of Alaska Anchorage on the "Exchange of Scientists" and "On Scientific Cooperation in Circumpolar Health."

- Ted Mala, M.D., director of the Institute for Circumpolar Health Studies, University of Alaska Anchorage, has established his new institute and continues his efforts to further international studies. In addition to continuing his work to develop cooperative research and scientific exchanges with the Siberian Medical Branch, Dr. Mala has extended his influence to the Far Eastern Branch and was successful in forging new agreements with the Far Eastern Branch and the Alaska DHSS.

- The Institute for Circumpolar Health Studies generously contributed to publishing Dr. Robert Fortuine's manuscript, "The Use of Medicinal Plants by the Alaska Natives," in this issue of *Alaska* Medicine.

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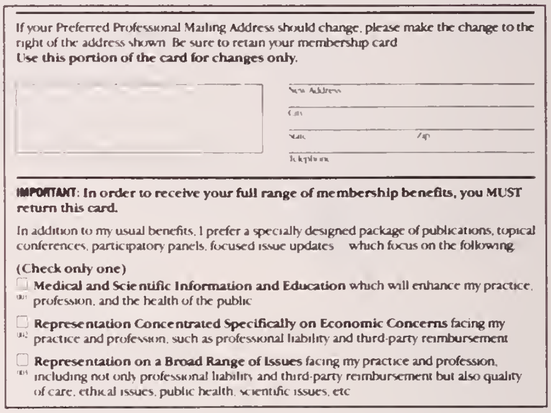

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President's Page

?

The flourishing malpractice field draws support from many sources: the oversupply of lawyers, judges whose decisions progressively widen the legal basis for malpractice suits, and juries which seem to feel that if someone is injured he should be compensated whether anyone is really to blame or not. These are only a few of the sources of support for malpractice, and like most, they are ones over which we as physicians have little direct control. We must look to the legislature or to the voting public to put into law measures which will slow this malignant growth that in some areas threatens to choke off the practice of medicine. The resounding victory of Proposition #2 in the recent election is a heartening instance of the success of this approach to dealing with the problem. The disadvantage of the legislative solutions and solutions which depend on initiatives on the ballot is that they are slow, expensive, and the outcome is uncertain.

There is, however, one important cause of malpractice litigation which we can bring under control, to a degree at least, at no cost to ourselves except for a little of our own time. These are the suits filed by patients whose anger is aroused, not so much by the quality of the medical care that they received, but by the feeling that they were not treated with concern and respect. When this happens, the slightest ripple in the course of their medical treatment may be welcomed by the angry patient as an excuse to go visit his attorney.

Sometimes patients become angry simply because they are irritable people who are looking for an excuse to get mad. At other times the patient's anger is justified because we have become so enamored of our technology that we sometimes forget that there is a person attached to the other end of the machine in our office. That person has expectations of his doctor which no amount of lab tests and x-rays can satisfy. If these expectations are not met, the patient feels demeaned and resentful.

What does a person want when he comes to the doctor? I think he expects three or four things. He wants someone who is a knowledgeable and sympathetic listener, someone who will explain to him what is wrong and what to expect from his illness, someone who will provide relief of the symptoms which brought him there. The doctor who combines compassion with competent medical care exemplifies that often used but seldom defined expression, "The art of the practice of medicine." The physician who deals with his patients in this manner is certainly not immune to suit. However, he will have few patients looking frantically for some excuse to sue him.

On the other hand, if the physician's attitude implies that he has not the time to listen or to explain, or that the patient is too dumb to understand anyway, he can rest assured that there is a storm brewing on his malpractice horizon. The patient so treated will find someone who will take the time to listen carefully to his story. His attorney will be happy to do so.

The malpractice problem has to be approached from all possible angles if it is to be contained. The ultimate solution will, no doubt, be a legislative one. In the meantime we can reduce the size of the problem significantly by the respect and understanding with which we administer care to our patients.

Irvin A. Rothrock, M.D.
President
Alaska State Medical Association

Auxiliary News

Alaska State Medical Association

The state auxiliary awarded two $1,000 scholarships in 1988. Heather M. Tonga from Cantwell and Mike Long from Ward Cove received them. A person must be from Alaska and pursing an education in some aspect of medicine in order to apply.

Heather is attending Duke University Medical Center. She is working toward a Bachelor of Medical Science degree and will graduate as a physician assistant in August of 1989. She plans to work in rural Alaska when she returns.

Mike Long is in his second year of the WAMI program at the University of Washington School of Medicine. He plans to specialize in internal medicine or obstetrics and gynecology. We wish the best of luck to both of them.

If you know of anyone who could use some help with their medical education, have them contact Linda Sutherland of the Scholarship Committee.

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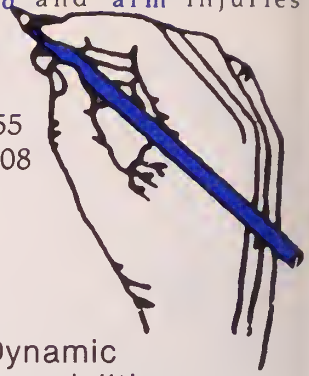

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References:

1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
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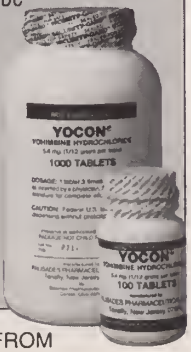

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Editorial

Little over five years ago few other than dedicated arctic enthusiasts had heard about arctic research, circumpolar health, or a national arctic research policy. International cooperation, hoped for by most, brought to mind the middle east and many other places on the globe that all share at least one characteristic—a winter temperature about 100 degrees hotter than the Arctic.

But how things have changed! In a short span of five years, interest in the Arctic has soared, reaching heights not seen since Peary and Cook raced each other to the North Pole in 1909. But while public excitement and interest are fickle, current scientific interest in the Arctic is likely to be steadfast and relentless.

Attention has focused on arctic people and in arctic science. Arctic people can teach the scientific community lessons of great value. And science can offer extraordinary benefits to arctic people. One of the greatest potentials lies in opportunities for international arctic research and cooperation.

Elsewhere in this issue of *Alaska Medicine* is a feature, "News from the American Society for Circumpolar Health." The society reports on just a few of the activities now underway to increase arctic health research, improve health services, and stimulate international circumpolar relationships. One of the leaders in increasing international circumpolar health research is Dr. Ted Mala, whose activities have taken him throughout the Arctic in pursuit of these goals. Building on the foundation laid decades before, Dr. Mala has seen tremendous successes arise from his seemingly endless supply of personal and professional energy.

As arctic research increases, so the need increases for publication and dissemination of findings. The Alaska State Medical Association has made a commitment to meet this need and was joined in this effort by the Alaska Public Health Association and the American Society for Circumpolar Health. With financial support of the Institute of Circumpolar Health Studies, University of Alaska Anchorage, of which Dr. Mala is Director, Dr. Fortuine's manuscript was able to be published in its entirety in this issue of *Alaska Medicine*. We are grateful for the opportunity to participate with the Institute for Circumpolar Health Studies in publishing this issue of *Alaska Medicine* and appreciate its support.

Our efforts to stimulate interest in the Arctic are beginning to pay. We must work together so that arctic people and arctic science can achieve new benefits for all.

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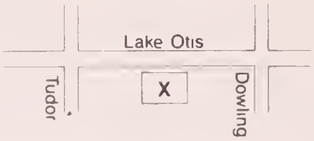

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Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined. However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted.

Pregnancy: Teratogenic effects. Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4.7%).

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

OVERDOSAGE

There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose. Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION

The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED

CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088-1712-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49). Light pink scored oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by C's on the other. Issued 1/87

Reference:

1. Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987;9(4):395-399.

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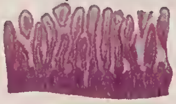
Aspirin and other nonsteroidal anti-inflammatory drugs weaken mucosal defenses, which may lead NSAID users to become prone to duodenal ulcers[1] For those NSAID users who do develop duodenal ulcers, CARAFATE® (sucralfate/Marion) is ideal first-line therapy. Carafate rebuilds mucosal  defenses through a unique, nonsystemic mode of action. Carafate enhances the body's natural healing ability while it protects damaged mucosa from further injury. So the next time you see an arthritis patient with a duodenal ulcer, prescribe nonsystemic Carafate: therapy for the ulcer-prone patient.

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Please see brief summary of prescribing information, and reference on adjacent page.

kiier being examined for effects of high-altitude cerebral edema.

ARMY RESERVE MEDICAL PROFILE NO.7

ALLAN J. HAMILTON, M.D.

Neurosurgical Resident and Research Fellow,
Massachusetts General Hospital, Boston, Massachusetts.
Captain, U.S. Army Reserve.

EDUCATION Ithaca College, B.A. (Magna Cum Laude); Hamilton College (Pre-med); Harvard Medical School.

RESIDENCY General Surgical Internship. Neurosurgical Residency, Massachusetts General Hospital.

CONTINUING EDUCATION Neurology and Neurosurgery Research Fellowship Training, National Institutes of Health.

OUTSTANDING ACHIEVEMENTS Olsen Memoria Fellowship, National Masonic Medical Research Foundation Albert Schweitzer Fellowship, International Albert Schweitze Foundation; Harvard Medical School Cabot Prize for Best Senior Thesis; recently published article, "Who Shall Live and Who Shall Die" in Newsweek Magazine.

"The work I'm doing in the Army Reserve fits perfectly with my academic research interests in civilian life. The Army is very concerned with the effects of high-altitude cerebral edema, which is a mirror model of cerebral hypoxia, something I deal with every day in our neurosurgical intensive care unit. I couldn't ask for a smoother transition. And that's true for a lot of Reserve physicians. All we really do is change our clothes, not our mindset.

"Some of the projects the Army is undertaking are on the cutting edge of research. For example, I'm currently involved in developing for the Army a prototype of a non-invasive intracranial pressure-monitoring device that we hope will allow us to measure pressure changes as the brain swells—without drilling holes in the skull. If we can get our design to work, such a device could revolutionize high-altitude medicine as well as civilian neurosurgical care.

"The quality of medicine and the caliber of people I've been associated with in the Army Reserve are, without question, equal to civilian hospitals. In fact, I'm giving serious consideration to applying for an active duty academic position in Army Medicine when my residency ends at Massachusetts General."

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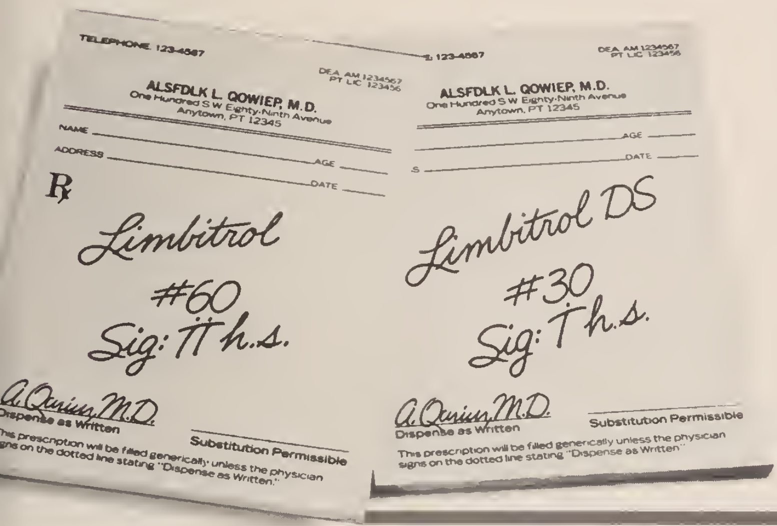
- 50% greater improvement with Limbitrol in the first week than with amitriptyline alone[2]



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One Hundred S W Eighty-Ninth Avenue
Anytown, PT 12345

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DATE ______

℞

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#60
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A. Quriuz, M.D.
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Substitution Permissible

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DATE ______

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#30
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Limbitrol® DS

Each tablet contains 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) Ⓒ

References: **1.** Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Feighner VP, *et al: Psychopharmacology 61:*217-225, Mar 22, 1979.

Limbitrol® Ⓒ
Tranquilizer—Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants; concomitant use with MAOIs or within 14 days of monoamine oxidase inhibitors (then initiate cautiously, gradually increasing dosage until optimal response is achieved); during acute recovery phase following myocardial infarction.

Warnings: Use with caution in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur when used with anticholinergics. Closely supervise cardiovascular patients. Arrhythmias, sinus tachycardia, prolongation of conduction time, myocardial infarction and stroke reported with tricyclic antidepressants, especially in high doses. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations. Consider possibility of pregnancy when instituting therapy.**

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: Use cautiously in patients with a history of seizures, in hyperthyroid patients, those on thyroid medication, patients with impaired renal or hepatic function. Because of suicidal ideation in depressed patients, do not permit easy access to large quantities of drug. Periodic liver function tests and blood counts recommended during prolonged treatment. Amitriptyline may block action of guanethidine or similar antihypertensives. When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady-state concentrations of the tricyclic drugs. Use of Limbitrol with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Should not be taken during the nursing period or by children under 12. In elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: Most frequent: drowsiness, dry mouth, constipation, blurred vision, dizziness, bloating. Less frequent: vivid dreams, impotence, tremor, confusion, nasal congestion. Rare: granulocytopenia, jaundice, hepatic dysfunction. Others: many symptoms associated with depression including anorexia, fatigue, weakness, restlessness, lethargy.
Adverse reactions not reported with Limbitrol but reported with one or both components or closely related drugs: *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke. *Psychiatric:* Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania, increased or decreased libido. *Neurologic:* Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns. *Anticholinergic:* Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue. *Endocrine:* Testicular swelling, gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Other:* Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. Withdrawal symptoms also reported with abrupt amitriptyline discontinuation. Therefore, after extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

Overdosage: Immediately hospitalize patient. Treat symptomatically and supportively. I.V. administration of 1 to 3 mg physostigmine salicylate may reverse symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

How Supplied: *Double strength (DS) Tablets,* white, film-coated, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt), and *Tablets,* blue, film-coated, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

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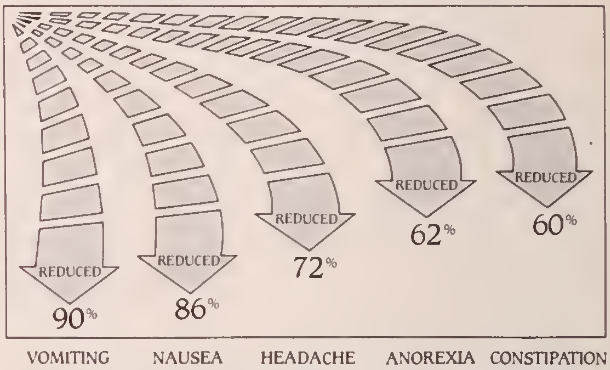
Caution patients about the combined effects of Limbitrol with alcohol or other CNS depressants and about activities requiring complete mental alertness, such as operating machinery or driving a car. In general, limit dosage to the lowest effective amount in elderly patients.

Percentage of Reduction in Individual Somatic Symptoms During First Week of Limbitrol Therapy*



*Patients often presented with more than one somatic symptom.

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